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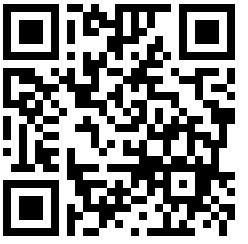

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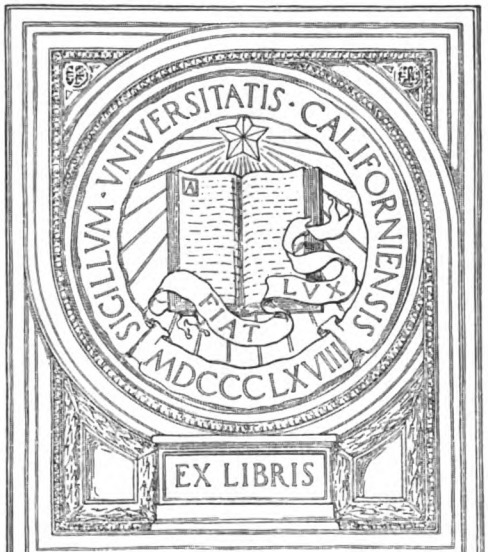


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THE

# CLINICAL JOURNAL

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW

*A WEEKLY RECORD OF CLINICAL MEDICINE AND SURGERY, WITH THEIR SPECIAL BRANCHES*

**IN TWO VOLUMES ANNUALLY**

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## A CLINICAL LECTURE

ON

# ACRO-PARÆSTHESIA

Delivered at St. Bartholomew's Hospital.

By J. A. ORMEROD, M.D., F.R.C.P.
Physician to the Hospital.

GENTLEMEN,—The complaint which has been dignified with the name of "acro-paræsthesia," appears at first sight to be rather trivial, still there are reasons for drawing your attention to it. For it is a very common complaint, yet not, I think, described in your text-books; it may be distressing and alarming to those who suffer from it; its nature is not completely understood, and it seems to be related to other obscure and interesting affections.

"Acro-paræsthesia" is a group of symptoms rather than a disease, but these symptoms are remarkably definite and constant. They may be described as follows:

(1) There are abnormal sensations, numbness, tingling, pins and needles, in the hands and arms, or (much more rarely) in the feet. Hence the name.

(2) There is pain in the same parts.

(3) Along with the numbness and the pain there is a feeling of uselessness in the hands and arms, enough to suggest to the patient the fear of paralysis, though there is no real loss of power.

(4) Though objective symptoms are as a rule conspicuous by their absence, yet it would seem that sometimes the hands turn pale, or turn red, or become swollen; and in some cases there is a certain slight impairment of sensory power in the fingers.

(5) These symptoms are paroxysmal, and their special time of incidence is the night time, though they may persist during the day, and thus become continuous.

(6) They are much more common in women

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than in men, in my experience almost confined to women, but undoubted cases have been recorded in men.

There was a nurse some time ago warded in John whose case I may take as an illustration of this affection.

She was a young woman, quite healthy in appearance except for slight anæmia, who stated that for the last three years she had felt numbness of the fingers and hands on waking from sleep. This she attributed at first to sleeping with her arms folded. During the last two or three months she had felt "run down," and the more so when she had been on night duty, as she was for the week before her admission. For the last six weeks she had noticed, on waking up, the numbness in the middle fingers of both hands, and during the last week this numbness had been succeeded by a cramp-like pain extending from the fingers of the left hand up that arm and round the shoulders to the right hand and arm. There had been several such attacks, always occurring as she woke from sleep, and the night before admission (November 15th) three attacks followed in succession, each worse than the other, and causing faintness.

She could not connect these attacks with any special kind of work. They came on after sleep, whether she slept at night as in the ordinary way, or whether she went to bed in the day, as when she was on night duty. But the numb feeling in the hands was now felt in the waking hours also. She had noticed no change in the appearance of the hands at the time of the attack.

She was admitted November 16th: Physical examination showed no evidence of organic nervous disease. The most that could be discovered was that she did not feel either a touch or a prick quite so acutely in the middle fingers (the fingers where the numbness was worst) as elsewhere.

Treatment has been very simple, viz. rest in bed, iron and nux vomica out of a mixture containing bicarbonate of soda and calumba; and with this she soon began to improve. The pain, indeed, disappeared from the time of her admission; but the numbness at night remained. Applications of the constant current (5 to 10 milliampères) down the arms were begun on November 22nd, and by December 6th she was able to leave the hospital apparently quite well.

I may compare this with a case which I saw many years ago. This was a woman between 50 and 60, active and healthy in most respects, but for eighteen months she had been troubled with the following symptoms. Every night she woke with pain in the fingers, hands, and up the forearms. The hands seemed to become stiff and useless, and when she got up she said they looked as if they were dead. The pain was severe, and she could not get to sleep again with it. She had some osteo-arthritis, as evidenced by the presence of Heberden's nodes on some of the fingers. She was a domestic servant, and ascribed her complaint to the use of water for scrubbing the floors; on this account she gave up her place, and after this her hands improved.

All cases are very similar in their main outlines, but there may be variations in detail: thus instead of the usual bilateral affection of the hands and arms, one hand only may be affected, and in some few cases the feet; Weir Mitchell alludes to cases in which the distribution is hemiplegic.

The paroxysmal character of the complaint, and its special liability to occur at night, or (to speak strictly) in connection with sleep, is an interesting point, and one which may often be noted in other diseases, for example asthma, gout, epilepsy. An epileptic boy who attended under me for many years at Queen Square at first had fits in the night only; subsequently he complained that they occurred in the daytime also, but on inquiry I found that the fit only came in the day if he had happened to go to sleep.

But curiously enough some patients with acro-paræsthesia complain that the symptoms come on in the daytime, when they make an attempt to use their hands for work.

The literature of this subject is not very large, and you may like to have a few references. Nothnagel wrote the first paper on the subject in the 'Deutsch. Archiv für klin. Med.,' 1867. In America, Putnam published twelve cases in the 'Archives of Medicine,' vol iv, 1880; and Sinkler wrote in the 'New York Med. Journ.,' July, 1881. I published a paper in the 'Reports of St. Bartholomew's Hospital,' vol. xix, 1883, which I believe was the first one written on the subject in this country; but I must mention that the late Dr. Steavenson, then physician in charge of our electrical department, had similar cases under his observation, as you will see from p. 244 of the same reports.

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Saundby wrote on the subject in 1885 ('Lancet,' 1885, vol. ii); and Schultze, writing in the 'Deutsche Zeitschrift für Nervenheilkunde,' 1880, gave to the complaint the name of "acro-paræsthesia." Since then several authors have contributed to the subject, as Laquer, Bernhardt, Dejerine, and others abroad, and in this country Savill ('Lancet,' 1901, vol. i), and Batty Shaw ('Practitioner,' 1903).

The circumstances which favour the development of the complaint seem to be these.

(1) *Sex.*—It is far more common in women than in men.

(2) *Age.*—Between twenty and fifty years of age is given as the commonest time of life for its occurrence. Probably these limits might be extended; but we may at least say definitely that it does not occur in childhood.

(3) *Occupation.* — The patients often connect their symptoms definitely with over-use of the hands in the daytime, particularly if this be combined with constant immersion in water, as in scrubbing or laundry work. Much needle-work may have the same effect. But I do not know that such a local origin can always be discovered. The nurse with whose case I began cannot think of any special employment which started her complaint.

(4) *Constitutional conditions.*—Probably anæmia, dyspepsia, and many other states of debility and depression may conduce to its development, but I do not think that it is invariably connected with any one of them. It would be a great mistake, I think, to ticket the symptoms as "hysterical," for they do not, so far as I know, occur in true hysteria, neither are the patients who suffer from them hysterical subjects as a rule.

The pathogenesis of the disease is not yet certainly known. Nothnagel considers it to be due to a vaso-motor spasm, and he forms this opinion on certain facts which he observed, viz. a pallor of the hands during the attack, a lowering of the surface temperature, the influence of cold in producing the symptoms, and of warmth in allaying them. This would bring the complaint into close relation with Raynaud's disease, which is also paroxysmal in its manifestations and in which pains and paræsthesia may occur.

But it is difficult to see why such a vaso-motor spasm should occur while the patient is asleep and warm in bed; and it seems, too, that a change of colour in the hands by no means always occurs; and that therefore such vaso-motor spasm is not an essential phenomenon, any more than it is essential to an epileptic fit, although the epileptic may change colour at the onset of a fit.

Nothnagel mentions some cases in which the hands became red and swollen, and Savill gives a case in which the symptoms of acro-paræsthesia were combined with the rare condition known as "erythromelalgia," in which a limb becomes painful, red, and swollen; such observations suggest a vaso-motor paresis, but they are. as a matter of fact, quite exceptional.

Indeed, the symptoms in the majority of cases suggest interference with the nerve supply rather than with the vascular supply. The numbness and uselessness are just what occurs after pressure on a nerve trunk; and as we have seen, pressure may be the first explanation that occurs to the patient. Mere pressure will not explain the pain, nor the bilateral distribution of the symptoms; still it is possible that there may be some analogous failure of nerve power of which we do not know the cause or the seat. No particular nerve trunks have been identified with the pain and numbness, so that we cannot call the complaint a neuralgia in the strict sense of the term. Dejerine and others have, indeed, found in some cases an incomplete analgesia corresponding in distribution to certain spinal nerve roots,* but I much doubt whether in the majority of cases any definite objective loss of sensation can be made out at all. So that we cannot definitely say what is the nature of the morbid process, nor yet what is the agent that originates it. It has been suggested that a "toxin" is at work, manufactured by the patient's own organism; but this is quite hypothetical.

As to the treatment of this complaint, I have generally found that rest from the work which has induced the symptoms (if this can be identified) coupled with simple general remedies, such as iron if there be anæmia, gentian and rhubarb mixture if there be dyspepsia, will do good. If these be not enough, then a nightly dose of potassium bromide should be given. Galvanism to the arms and hands is another most excellent remedy. And I think that thus a cure may be expected. Perhaps the cases that I have seen may have been mild

* Dejerine and Egger, 'Revue Neurologique,' January 30th, 1904.

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ones, for I notice that some of the foreign authors take a less hopeful view of the matter. Nevertheless one is justified in re-assuring the patient, who is often haunted (perhaps not unnaturally) by the fear of an impending paralysis, and in telling her that no serious results will follow.

But in this connection I must make one reservation. Before giving any such favourable prognosis you must make sure that you are dealing with an "idiopathic" acro-paræsthesia, and not with a case where numbness and pain are the symptoms of underlying organic disease. In an out-patient whom I saw at Queen Square, pain and numbness in the upper limbs were followed by wasting of the hand muscles with electrical reaction of degeneration; the disease was a peripheral neuritis, and not a mere neuralgia. Disease of the cervico-dorsal nerve roots may produce similar symptoms. Numbness and pain may occur in certain progressive degenerations of the spinal cord, such as tabes. Thus a careful examination of the nervous system, to exclude such conditions as these, should be made before you venture on any prognosis. You must remember, too, that numbness of the extremities is sometimes due to Bright's disease. Dr. Batty Shaw gives a striking instance of this. A man was admitted to University College Hospital, complaining of this particular symptom, and in a day or two afterwards uræmia set in, of which he died.

*April 20th*, 1908.

**Non-fatal Serum Reaction.**—A. P. Ohlmacher reports two cases of severe reaction to therapeutic serum injections. In both cases the contingency of hypersusceptibility had been considered and attempted to be met by administering several "immunising" injections during the first eight or ten days of what is shown by animal experimentation to be the sensitising interval. In both cases, the more common "accelerated reaction" of the serum disease, as defined by von Pirquet and Schick—*i.e.* urticaria, joint pains, and fever—failed to appear. It seemed as if these more usual manifestations accumulated and took the form of one grand explosion. Ohlmacher says the good results following the injection of horse serum in chronic rheumatism are due to the periodic hyperæmia in the affected joints by the "accelerated serum reaction."—*Med. Rec.*, vol. lxxiii, No. 13.

# GONORRHŒA IN THE MALE, WITH SPECIAL REFERENCE TO TREATMENT.

A Clinical Debate held under the auspices of the Chelsea Clinical Society.

**DR. J. H. DAUBER, President, in the Chair.**

MR. J. G. PARDOE, in opening the discussion, said he conceived that it was not the function of the introducer of a debate to deal largely in matters of detail, but to introduce a kind of skeleton, upon which clothes might be hung. But such clothes might possibly be changed or altered during the debate, and something more elaborate and beautiful might be substituted for the garments which he had placed upon it. Those who arranged the debate had wisely decided that it would be better for one speaker to take one part of the subject, and another speaker another part. In that way one could better bring forward points which were or might be debatable. In any debate it was well to take the subject in an orderly manner. It was his function to deal with the acuter symptoms of urethritis.

In the first place it was necessary to consider the prophylaxis of gonorrhœa, not by means of the obsolete and departed Contagious Diseases Act, but by more modern methods which had been introduced into other countries, but not with so much success in ours, owing to public opinion. In the German Navy an important means had been adopted to prevent the occurrence of gonorrhœa. It probably started first with the defence of Pekin and its relief by the allied forces. Gustav Tandler, a medical officer in the German Imperial Navy, went to Pekin with the allied forces in 1903. Two hundred sailors of the Embassy Guard were returned by him to Europe. Of those 200 sailors, who had done three years' service, 40 per cent. had suffered from primary gonorrhœa in China. They had been treated in the ordinary way after the disease had well developed. Tandler had a fresh draft of 170 men under his medical care, and they were under him for three years. He introduced a course of compulsory prophylaxis, by which the men were obliged to report themselves, not only on the first

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symptom of venereal disease declaring itself, but also immediately after sexual intercourse, under heavy penalties if they neglected to do so. Those 170 men underwent 1560 prophylactic treatments. Of those 1560, fifteen men only got gonorrhœa, which worked out at 1·6 per cent. in 170 seamen. Those men were instructed to report immediately after intercourse, or as soon as possible, and they were injected in the anterior urethra. By that he meant only that portion of the urethra which lay in front of the inferior layer of the triangular ligament in front of the compressor urethræ muscle, *i. e.* the bulbous and penile urethra. The injection was usually 10 per cent. protargol in an aqueous medium or in petroleum jelly. One, or at most two, injections were given after intercourse, and the results were as he had described. It was not necessary to further elaborate the statistics of prophylaxis in the German Navy, it was probably sufficient to say that certain German naval officers had stated that the admissions of liberty-men for venereal disease had in the last three years dropped, at a very moderate estimate, 50 per cent. That was striking, but it was hardly to be hoped that, with public opinion as it was in this country, it would be possible in the public services to institute compulsory prophylactic treatment. And it was a point which was capable of debate from one side or from the other, whether the fact of minimising the perils incident to illicit intercourse would be an incentive to immorality, or the reverse. But their business as surgeons and physicians was not so much to consider the morals of their patients as to prevent disease or cure disease, and if by any means that could be done that means was probably justifiable. For that reason he had touched on prophylaxis. He next passed to the actual treatment of acute gonorrhœa. That seemed to fall naturally under the headings of (1) abortive treatment, (2) modified abortive treatment, and (3) expectant treatment, the last of those being the most old-fashioned. By abortive treatment he meant absolutely cutting short the disease when infection had already taken place, before it had had time to get even to the so-called acute stage of gonorrhœa. And for pure abortive treatment it was useless to attack the patient after more than twenty-four, or at most forty-eight, hours had elapsed from the beginning of the symptoms. In other words, to secure true abortion of gonorrhœa, one must have the patient under care when the symptoms simply consisted of slight itching of the meatus, with, perhaps, slight gumming of the lips and a little sero-purulent sticky discharge. After acute yellow or yellowish-green discharge had commenced, it was of no use hoping to be able to treat it by the abortive method. What methods were there at the hand of the physician and surgeon to secure that true abortion of gonorrhœa? There were several. He desired to remind his hearers of the mode of infection and spread of gonorrhœa along the urethra. It commencd at the meatus and spread by direct continuity in the epithelium of the mucous membrane. And in the first four or five days, unless the man was peculiarly susceptible to the gonorrhœal poison, the infection was limited to the first inch or two of the urethra. That fact anybody could prove by cocainising or otherwise locally anæsthetising the urethra and examining it with the electric light by means of the urethroscope, when he would see that the blush of inflammation was limited to the first inch or two of the mucous membrane. That gave the clue to the abortive method, which could be used by those who were skilled with the urethroscope, by painting through a tube the whole of the mucous membrane from a point a little behind that which was obviously infected and red, forwards to the meatus. The same result could be secured by those who had not a urethroscope, or who were not skilled in its use, by limited anterior urethral injections. In those cases where abortive treatment was carried out by limited injections, the urethra should be blocked by pressure of the finger and thumb at the peno-scrotal angle. It was not required that the injections should go into the bulbous urethra. He intended only to direct attention to the broader aspects of the subject, and he did not, therefore, detail the large number of substances which could be employed for the purpose. In his own opinion, protargol and silver nitrate were the best agents, and they could be used in considerable strengths if the mucous membrane were previously lightly anæsthetised: *i. e.* 10 per cent. protargol, or 2 to 4 per cent. silver nitrate. The method itself was very simple. The injection should be given as soon as possible. It should not be repeated the next day, but the day after that. If the case was going to be a successful one, the probability was that those two

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injections in a period of forty-eight hours would be sufficient. The disease was cut short, and nothing further followed. The other method was by means of anterior urethral irrigation—less severe, probably equally efficacious. The agent used was generally permanganate of potassium in a strength of 1 in 1000, and the irrigation should be given with the same restrictions as to the stopping of the backward flow as already mentioned, and given night and morning for at least five consecutive irrigations.

Next, he wished to allude to the class of case where the discharge had already become well established, in which there was the usual penile pain, the scalding micturition, the painful erections at night, and all the other classical phenomena of gonorrhœa well in evidence. In such a case as that the pure abortive treatment was useless, and the practitioner had to decide between the two methods—the expectant method, and the modified abortive method. There could be no question that anterior irrigation in those cases, or a large proportion of them, was exceedingly successful in causing a complete cure in certainly half the time that the expectant treatment would. But in a certain proportion of cases, where, perhaps, the disease could not be cut short very much by irrigation, it could be rendered very much less unpleasant than usual where the patient had to walk about in following his business, with his urethra discharging freely, causing great pain and discomfort. Moreover, by well carried out modified abortive treatment, the risks of the backward spread of the disease to the bulb, the membranous urethra, the prostatic urethra, and the urethral appendages and testicles was very much limited and minimised. He must be excused from going into details. The best agent was usually permanganate of potassium, and in cases of acute gonorrhœa, where the urethra was tender and sore and where manipulation was, therefore, distinctly unpleasant, the strength of the solution should be reduced at the start. To begin with, a strength of not more than 1 in 5000 should be used, worked up by gradual increments of 1 in 500 to a maximum strength of 1 in 1000. Irrigation should be given twice a day, and the whole course should last ten days to a fortnight. It was of no use to miss one or two of those irrigations; if the patient wished to have the best effect he must adhere to the treatment all through. The irrigations should not be given by a catheter; no instrument should be passed down the urethra, but they should be given by means of the ordinary irrigator can holding two to three pints, suspended one or two feet above the patient, who is lying recumbent on a couch. The nozzle, which should be blunt, should just fit the urethral meatus and not enter more than half an inch. By gently blocking the meatus with that blunt nozzle, and alternately blocking and relaxing the part, one could balloon the anterior urethra back to the bulb, if it was thought the inflammation had got as far as that, or stop it at any point in its backward course. By that method one could flush out the portion diseased by means of the urethral irrigator and nozzle. The reason for the avoidance of passing instruments down the urethra was that, however gentle the doctor might be, in the inflamed condition of the mucous membrane there was the risk of peri-urethral abscesses being caused, or the risk of slight lacerations leading to indurations, which might eventually become strictures. In a considerable proportion of cases the result was excellent; the discharge in two or three days disappeared, and though, if the treatment were stopped at that stage, the discharge might come back in its original fury, yet if persisted in the discharge did not return, and the result was that the urethra soon became dry and remained so. That was the bright side of the picture. The other side was that discharge sometimes returned three or four days after the termination of the course of irrigations. Those who were wise would, when instituting the treatment, warn the patient that having come when the disease was thoroughly well established, abortion could not be guaranteed, but there was a good chance that the disease would be shortened in its course, and it could be promised that the disease would be less unpleasant in its incidence.

The other method was the expectant one, and it was right that he should mention certain points which were distinctly of interest in discussing the two methods, the drawbacks of the one and of the other respectively. The drawbacks of the modified abortive treatment lay largely in the question of the detail used in carrying them out. If one was so unfortunate as to drive back the gonococcal pus into the deep urethra in trying to abort an anterior urethritis, one certainly ran the risk of setting up

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acute prostatitis, vesiculitis, and epididymitis; and patients who had, perhaps, had many attacks of gonorrhœa cured before by simple means, without unpleasant complications, if treated by the new method, and they had epididymitis or other complications, would never be persuaded that the practitioner was not at fault. Therefore, one should be very careful to limit the treatment to the anterior urethra, and one should never allow the patient to carry out the treatment on himself. On the other hand, there was the expectant method of treatment—the old-fashioned method, by alkalies, and sedatives, such as hyoscyamus, and by such drugs as buchu. That method certainly in a large number of cases had most excellent results; and, although with the expectant treatment susceptible individuals got the complications, yet his own opinion was that many of the complications were due to the improper use of the ordinary urethral injections by patients in the acute stages of the disease. That was one of the debatable points which he mentioned at the outset, and perhaps it was one of the garments which would be torn to shreds in the subsequent discussion and fresh clothes substituted. If he were going to treat a patient by the expectant method he would prohibit the use of injections absolutely until the acute symptoms had subsided, until the greenish-yellow, thick purulent discharge had become of a more milky consistency, until the acute urethral scalding was past—*i. e.* until about the third or fourth week of the disease. At that stage and onward he thought anterior urethral injections would be of benefit. He treated his patients with copious diuretics and alkalies. It was known that the gonococcus did not live in alkaline media. He gave urinary sedatives in large doses, such as hyoscyamus ½ to 1 drachm of the tincture, purgation, and above all, rest. If one could obtain rest for the patient it was half the battle. He had so far been speaking of acute anterior urethritis. Acute posterior urethritis was another matter altogether, and he desired to record his firm opinion that irrigation for acute posterior urethritis was totally unjustifiable. That was an opinion in direct contrast to the practical originator of urethral irrigation, namely Janet, who treated a large number of patients with acute posterior urethritis most successfully by posterior irrigation into the bladder. There were cases on record where posterior irrigation had been used in acute urethritis where cystitis, ascending uretero-pyelonephritis, and death had resulted. If that series of events occurred when the patient was being treated by the expectant method, blame was not cast upon the practitioner, but if he were so unfortunate as to have such a series of events when treating a case by posterior irrigation, it would be most difficult to avoid the conclusion that this very serious complication was due to the method employed, and the minor ills, such as acute epididymitis, occurred with too unpleasant a frequency in connection with posterior irrigation in acute posterior urethritis. Therefore, he treated his cases of acute posterior urethritis by the expectant method, by hot hip-baths, suppositories, and various alkalies and urinary sedatives, and, if necessary, by morphia.

Lastly, it was essential that he should mention something about the serum treatment of the disease and of the inoculation methods, which had met with a certain degree of success, particularly in the fulminant type of the disease. He would not weary the meeting with a recital of cases from his own practice, though he had had some striking ones, but would simply say that by means of the inoculation of vaccines certain cases of fulminant gonorrhœa, attacking rapidly the whole of the lower urinary system, the genital system, and the joints and eyes, had cleared with remarkable rapidity under the use of the gonococcus vaccine, and certain cases had apparently benefited by the use of polyvalent streptococcic serum, which had been used because it had been found that the gonococcus could not live in culture media impregnated with the polyvalent serum.

In conclusion he congratulated the Society on having chosen the subject for debate. No one could know better than the Chairman, who had much to do with the results of gonorrhœa in the female, or than himself, who had much to do with its results in the male, what those results were, and no one could feel more strongly the necessity for an improvement in the treatment of gonorrhœa in its acute stage, and for the prevention of the later complications, and for a better treatment of those complications when they did occur.

The CHAIRMAN said he was sure all present had listened with great interest to Mr. Pardoe's instructive address. He had dwelt on the extreme im-

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portance of gonorrhœa and its sequelæ, because it entered very largely into the medical man's practice. The results of gonorrhœa were often disastrous and far-reaching, affecting sometimes not only the present generation but the next, as in ophthalmia neonatorum. The subject of the prophylaxis of gonorrhœa was a very large and debatable one. It had been discussed in parliaments and councils, by the laity as well as by the profession, and there was still a great difference of opinion as to the measures which were appropriate and those which were not appropriate for the community to adopt. It was a vast question, and could not be properly gone into that evening; but an interesting phase of it was whether prostitutes should be allowed to solicit in streets and public places of entertainment, or whether, instead, houses under Government control should be tolerated and legalised. But those and other questions were really outside the scope of the present discussion, and whatever the private opinions of medical men there that evening might be, they would not, he feared, influence the course of legislation. An important point which Mr. Pardoe did not refer to, but which affected the question closely, was that the practitioner might, in his own sphere, point out to the laity the importance of circumcision. It was agreed in the profession that it was one of the best prophylactic measures that there could be against venereal disease. There could be no doubt that men who had been circumcised and had the skin over the glans penis hardened by constant exposure and kept dry, did not acquire gonorrhœa with the same frequency, if at all, as did men with a long prepuce and moist glans penis. That was probably admitted by everybody. And it was a common event nowadays for parents to have their male children circumcised, whether they had a long prepuce or not. And he thought that course might be advised with propriety by medical men as a useful and cleanly prophylactic measure against gonorrhœa and against syphilis. All German methods did not commend themselves to him, and the compulsory method mentioned by Mr. Pardoe as operative in the German services did not appeal to him favourably. He could hardly imagine an English medical man being willing to wait and hear the report of sailors and soldiers immediately after connection, and attend upon them within a short time and inject their urethras for them. It seemed very much like pandering to their immoral practices. They might use a little protargol themselves. He would not enter into the details of what Mr. Pardoe had brought forward, but would leave it to others there that evening who knew more about the subject. He had been glad to hear Mr. Pardoe touch upon serum therapeutics. He believed that sera and vaccines had a great future. On looking back and seeing how serum treatment had in many directions revolutionised treatment and results, he could not help looking forward hopefully to the time when a man having acquired gonorrhœa would at once go to his medical man and receive vaccine treatment. Rogers and Torrey, in America, had done much work in that direction, and spoke most favourably of it. Serum treatment seemed such a simple and cleanly method of dealing with an objectionable disease, that he hoped it would be possible to rely upon it.

Mr. Aslett Baldwin said: In the course of the admirable speech made by your President at the annual dinner of the Society he optimistically stated that a method would be arrived at in the course of this debate of immunisation against gonorrhœa. We have all, no doubt, noticed that when a person has had a number of attacks they tend to get less acute, so that immunity, more or less partial, is gradually developed. It not uncommonly happens that a patient appears complaining that he has ricked himself or tumbled over the coal-scuttle, and one finds on examination an acute epididymitis. In these cases, when very acute and painful, I have always found very rapid relief follow the administration of tartrate of antimony and sulphate of magnesia—one sixth of a grain of the former and a drachm of the latter in an ounce of peppermint-water every one or two hours, till they have either taken six doses or been sick; in either case they take no more. They are recommended to rest in bed with the testicles supported as high as the top of the pubic bone. Glycerine of belladonna is painted on the inflamed part and hot fomentations are applied; these are renewed every hour; equal parts of guaiacol and lanolin form a very useful ointment at this stage. As soon as the inflammation begins to subside equal parts of ung. bellad. and ung. hydrarg. Co. are applied, and the fomentations continued. When

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the patient is allowed up, the ointment is still applied and the testicles well braced up by a suspender. As soon as the first medicine is omitted the patient is given sodii salicylatis gr. x potassii iodidi gr.v three times daily, and, if necessary, a small dose of sulphate of magnesia before breakfast, followed by three quarters of a tumbler of hot water. As soon as the tenderness will allow of it, ung. hydrarg. Co. is rubbed into the induration in the epididymis daily, after washing with soap and warm water, and instruction given that if the skin becomes inflamed the ointment must be discontinued for a day or two. This massage is commenced as early as possible in order to get rid of the inflammatory exudation before it organises into fibrous tissue, causing blocking of the epididymis and sterility of the affected organ. Where this is not being accomplished to one's satisfaction the dose of iodide taken internally should be increased and a drachm of the liq. hydrarg. perchloridi added. I believe also the hypodermic injection of fibrolysin is useful, or its equivalent—thiosinamine and salicylate of soda. The soda is added to make the thiosinamine soluble. This comes out much cheaper than fibrolysin, though the latter is very conveniently put up in glass capsules, each containing one dose, *i. e.* 3 grains of each of the above two drugs in 40 minims of water. When using this drug I have generally injected it under the skin of the back every other day. During an epididymitis I generally allow the patient to use an injection, such as sulpho-carbolate of zinc, two grains to the ounce of water, in the anterior urethra four times daily, after passing water, and tell them to retain it for two minutes.

After the subsidence of an acute attack of gonorrhœa it is not uncommon for the patient to be troubled with a gleet or the passage of flakes and shreds in the urine. For successful treatment it is necessary to try and locate the parts where the inflammation persists, or the glands or follicles where gonococci may be lurking safe from the reach of irrigations or injections. The meatus should be held open and pressure made from behind forward to see if pus is squeezed out from small glands in that region; the finger should then be passed along the under surface of the penis and made to palpate the anterior urethra in search for thickenings or indurations. These can sometimes be felt by the patient himself when not perceptible to the surgeon's finger. The patient should then pass urine into three or four urine glasses, about two ounces into the first and about five ounces into the second. If the flocculi are mainly limited to the first, they point to trouble in the anterior urethra; if the urine in the second glass is cloudy and contains flakes, it points to mischief in the posterior urethra; especially is this so if the third and fourth glasses contain cloudy urine. Thin filmy flakes are probably from the anterior urethra, shorter and more solid ones from the posterior. Considerable quantities of muco-pus in the last glass would probably come from the bladder. If the anterior urethra is injected or irrigated with a coloured solution, such as permanganate of potash or methylene blue, and urine is passed afterwards, flakes from the posterior urethra will be unstained. In dealing with a lesion of the anterior urethra the most exact and scientific method is to introduce a warmed and lubricated urethroscopic tube containing its obturator, noting the position of any tender spot as the instrument sinks in by its own weight. The instrument should be lubricated with sterile olive oil and the meatus washed with a swab dipped into some antiseptic before the instrument is passed. I always take the instrument from warm cyllin lotion and wipe the meatus with the same. When the cannula has passed as far as it will go the left wrist is rested on the patient's groin and the cannula steadied by the left hand, while the obturator is removed; this is often best done by slightly rotating it at first; it must not be removed with a jerk, or the end of the cannula will hurt the patient.

A long, thin stick with cotton-wool twisted round its end, as is done on a Playfair's probe, is then gently passed to the bottom of the cannula and any oil present carefully mopped up; unless this is done no proper view of the urethra will be obtained. The door of the instrument should now be closed and some air passed in, and the opening in the triangular ligament examined and successively each part of the urethra is examined as the instrument is slowly withdrawn. In this way congested patches are seen, inflamed or obstructed glands, ulcers, granular patches, warts, and narrowings of the tube. By inflating and deflating it can be estimated if an inflamed part is lissom, showing the existence of superficial inflammation, or whether it

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is unbending and stiff, showing deep infiltration likely to lead to the formation of a stricture. Congested patches are treated by the application of wool twisted on the sticks already mentioned, dipped in solution of silver nitrate, commencing with 1 per cent. applied two or three times a week, according to the effect produced, and gradually increased in strength as may be necessary; in some cases up to 60 grains to the ounce, any excess being carefully mopped up. Liniment of iodine, carbolic acid, or 10 per cent. sulphate of copper solution are applied in the same way. I think the silver and the iodine are the most useful. Occasionally solid silver nitrate is applied to an obstinate granular patch, or they may be incised longitudinally and superficially, the subsequent scarring causing obliteration of these vessels. Iodine or mercury ointment rubbed into the penis externally to the patch will assist resolution. Obstinately inflamed and patulous glands are treated by electrolysis, about a 3 milliampère current for about twenty seconds, or until the gland becomes white; or the bent end of a fine probe armed with solid silver nitrate may be inserted into each with a fair prospect of cure. Retention cysts are best treated by electrolysis, or, failing that, by solid silver nitrate to their interior after incision. A less exact method of applying solutions to various parts of the urethra is to pass the cannula till it meets with a definite area of tenderness, then to stop its introduction at once and swab the part to remove any lubricant, and afterwards apply the selected solution, but without the aid of the rest of the urethroscope, and, therefore, without the aid of sight.

A discharge from the posterior urethra is treated by large irrigations with dilute antiseptic solutions, such as potassium permangante 1·6000, gradually increased in strength up to 1·1000, if no irritation is caused and improvement is continuous. The irrigations are preceded by massage of the prostate and seminal vesicles. To do this the patient should either stand with the body flexed at the hips to a right angle and the lower limbs somewhat separated, the arms resting on a support, or be in the knee-elbow position on a couch. The surgeon's index finger should be protected by a thin rubber glove, lubricated with vaseline. The hand and finger must be kept in the axis of the forearm, or it will be often impossible to reach high enough up the bowel. The massage is done gently and with the last phalanx. The prostate is massaged from each side towards the middle line, and then along the middle line backwards. The seminal vessels must be massaged very carefully, bearing in mind that their ducts are very small, and will only allow their contents to pass at a certain rate. Considerable pressure will at times have to be made by the flexed fingers of the surgeon on the patient's perineum before the index finger can reach high enough up the rectum. If progress is not satisfactory after this treatment has been applied daily for about fourteen days, instillations of nitrate of silver are injected into the posterior urethra twice a week by means of a Guyon catheter and syringe or an Ultzman syringe. The strength of the solution is at first 1 per cent. This is gradually increased up to 4 per cent. if it causes improvement. The end of the syringe is inserted within the anterior end of the compressor urethræ muscle; this is generally over five inches and under seven inches from the meatus, and can be felt to suddenly give way and admit the end of the instrument after gentle but persistent pressure. Ten or fifteen drops are instilled at a time. If much pain is caused, ten drops of 4 per cent. novocain solution may be injected into the posterior urethra a few minutes previously. If there is at the same time obvious discharge from the anterior urethra, about ten drops of the 1 per cent. solution may be distributed along the anterior urethra as the instrument is withdrawn. If the discharge from the posterior urethra persists in spite of this treatment, the prostatic urethra may be lightly cauterised from behind forwards two or three times through the prostatoscope with solid nitrate of silver, fused on a suitably strong wire, like a catheter stylet. The above instrument is a long urethroscopic tube with a shoulder near the end and a window on the convex side of the bend. The deep urethra will have to be anæsthetised to allow this instrument to be introduced. When it is in place it may be seen that the sinus pocularis, which opens on the summit of the verumontanum, is somewhat gaping, a stylet tipped at its point with nitrate of silver should then be passed for a quarter of an inch into the sinus.

If treatment of a chronic posterior urethritis appears to make the condition worse, or if it has not distinctly improved after about six weeks treatment, it may be well to leave off all local applications and give the

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patient general tonic treatment and change of air. It is well also to remember that the appearance of filmy flakes in the urine may be kept up by injections.

Inflammation of Cowper's glands is evidenced by tenderness and pain, and perhaps a palpable swelling to one side of the perineum just in front of the anus. When this is present only, the mildest injection must be used and only in the anterior urethra. The patient should be put to bed, the bowels kept well opened, and a hot-water bottle applied to the perineum. If the condition becomes chronic, iodine or mercurial ointment should be rubbed in over the gland. In some cases the gland has had to be excised to effect a cure. It is one of the causes of a chronic discharge from the urethra. If suppuration takes place, an incision should be made early to prevent burrowing of the pus.

An intermittent discharge from the urethra is caused by chronic seminal vesiculitis. In these cases there is more or less constant pain about the urethra, perineum, bladder, testicles, rectum, or it may radiate to the back, abdomen, inguinal regions, or the thighs. The finger in the rectum discovers the vesicles to be full and tender to pressure, and there may be inflammatory induration about them. In acute cases there is frequent straining micturition, and, in addition to the above pains, pain is referred to the hip or sacro-iliac joints of the same side and down the outer side of the thigh. The symptoms may simulate appendicitis. There may be priapism and emissions of blood-stained semen. A rectal examination is necessary for the diagnosis. The treatment consists of rest in bed with the pelvis raised, hot rectal irrigations with an apparatus having an in-and-out flow-tube, hot sitz baths, morphia and belladonna suppositories. Such drugs as salicylate of soda in 10 grain doses should be given every four hours, and if any urethral treatment is continued, it must only be very mild injections or irrigations, provided they cause no further irritation. As the disease becomes chronic the vesicles should be stripped by massage, gently enough not to cause pain, two or three times weekly, with a full bladder.

The treatment of acute prostatitis is much the same. If the pus forms a swelling posteriorly between the prostate and the rectum it should be let out through a semilunar incision in front of the anus, gradually deepened between the rectum and bladder, and good drainage provided. If the pus bursts into the urethra the cavity of the abscess should be induced to close as soon as possible, to prevent chronic suppuration and the formation of phosphatic calculi. When the degree of tenderness will allow of it, the prostate should be massaged to empty the cavity of pus, and the patient directed to pass water; the urethra and bladder are then irrigated with an antiseptic solution. This is repeated twice daily.

In chronic cases massage followed by irrigation are useful, also rectal injections of hot salt water at a temperature of 110° to 118°. The bowels should be kept regular, the diet should not cause the urine to be irritating, the general health should be kept as good as possible. The posterior urethra should be treated appropriately if necessary, and an ichthyol suppository placed in the rectum at night will be of service. The treatment naturally takes a good deal of time and perseverance.

My time is nearly up, but as a final word I should like to advocate the treatment of gonorrhœal rheumatism by daily injections into the rectum of 10 c.c. of polyvalent anti-streptococcus serum and the treatment of the inflamed joints, tendon sheaths, etc., by Bier's method of passive hyperæmia, the time occupied being up to twenty hours daily. Also massage and much earlier active and passive movement than is often used, splints being discarded if possible, or only put on at night to relieve pain and procure sleep. Salicylate of soda or salol are useful internally, also quinine, mercury and iodide of potassium. Scott's ointment with belladonna should be applied to, or rubbed into, the affected parts, when not too tender, the rubbing always being in an upward direction, and injections of thiosinamine used to prevent threatened ankylosis. When the condition is more chronic the part should be treated as described during the day, and at night, wrapped up in a piece of flannel soaked in a saturated solution of common salt and covered with a waterproof protective, and the patient encouraged to use the limb as much and as early as possible.

Dr. T. W. PARKINSON said that the aspect of the question which concerned him most, but which was only partially touched on in the introduction, was prevention. By devoting so much attention to the

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treatment of gonorrhœa he thought the medical profession was on the wrong lines. Great efforts should be directed to preventing the disease. He differed from the remark which had been made that there would be very little progress made in the prevention of the disease. If the profession were to take the matter up in earnest, a large reduction in the amount of gonorrhœa in the country would be affected, and in that way there would be a great diminution in the number of inmates of hospitals, and the poor-rate would be reduced, because a large number of people were incapacitated through having gonorrhœa. Men had acquired gonorrhœa, and for months afterwards had been practically useless. There was little doubt that if it had not been for a number of people with misguided ideas, there would have been legislation on the subject before now. It reminded him very much of what had been done for consumption by means of sanatoria all over the country, to take in and to treat isolated patients instead of making an effort to prevent or lessen the chance of occurrence. In regard to treatment, he was sorry that in consequence of pressure of time Mr. Pardoe had passed over very rapidly the question of rest in treatment. Mr. Pardoe knew quite well, probably better than he himself did, that if a patient had an attack of gonorrhœa and consulted his medical man at the beginning, if that patient could be put absolutely to rest many of the unfortunate sequelæ, such as orchitis, were prevented. It was a golden rule that the patient with gonorrhœa should have absolute rest. Failing that, there were other precautions which were sometimes omitted. If the patient had to walk about during his attack, it was very important that he should be braced up; he should have suspenders for his testicles, and should wear bathing drawers to prevent the penis working about much during walking. Those precautions and rest should play a far greater part in the treatment of gonorrhœa than they did at present. His object in speaking had been to emphasise the fact that, in his opinion, efforts were directed at the wrong end of the subject, and that particular attempts should be made to lessen the incidence of the disease in the community.

The CHAIRMAN explained that he did not mean in his remarks that he thought the medical profession should keep quiet on the matter (as a class he thought that medical men were far too quiet on the subject; it was a failing of the medical profession, especially lately, that they kept their opinions too much to themselves and omitted to instruct the public sufficiently, not only on the matter they were discussing, but on hygiene and kindred topics), but he felt that prophylaxis was rather too large a subject to discuss that evening. He desired to ask Mr. Pardoe whether he had discarded the syringe altogether in the treatment of the disease. Was the medical man to advise his patient to throw his syringe in the fire? Did Mr. Pardoe consider continuous urethral irrigation to have entirely superseded the syringe? For himself he favoured the copious irrigation method preferably to the syringe.

Dr. J. M. A. COSTELLO, Galway, said he wanted to say a word about the vaccine treatment of gonorrhœa. That treatment did not concern so much acute gonorrhœa as chronic. Its use in acute gonorrhœa, indeed, had been more or less disastrous. At the same time there were also some disastrous results from its use in chronic gonorrhœa. Very good results in chronic gonorrhœa had also been achieved from its use. A gentleman went on his holiday and came back with a marked attack of gonorrhœa, and it developed into a chronic condition, so that life was a misery to him. He was also very much depressed and neurasthenic, and regarded life as not worth living. He applied to a medical man to treat him, and that gentleman made unsuccessful attempts at local treatment. Then he was put under vaccine, stipulating that while vaccine treatment was being carried out local treatment should be applied also. After the first injection no very good result followed. A week later another injection was given, and although the discharge was not lessened very much, the patient said he felt better in himself, better than he had been for a considerable time. The medical man encouraged him, and a week later gave him another injection. That cleared away all the discharge, and on examination of the discharge there were very few gonococci in it. A fourth injection was given, and all the discharge stopped. The case was not his own, but he knew of it, and thought it might be interesting to the members to hear of it.

The CHAIRMAN expressed surprise at Dr. Cos-

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tello's remark that anti-gonococcic sera were only useful in chronic gonorrhœa, because Robert Taylor, Consulting Genito-urinary Surgeon to the Bellevue Hospital, New York, in the 'American Journal of Obstetrics' for January this year, stated that those vaccines seemed useful in the active and nascent conditions of gonorrhœa when the gonococcus might be acted upon, but were impotent and inert in chronic cases in which the activity of the gonococcus was impaired. Dr. Costello's case therefore corrected the impression of Dr. Robert Taylor on the point, and indicated clearly that anti-gonococcic sera were useful in both chronic and acute conditions of gonorrhœal urethritis.

Dr. MANSELL MOULLIN asked whether Mr. Pardoe had tried treatment by means of anti-streptococcic serum, and whether that was the vaccine to which allusion had been made.

The CHAIRMAN said that Robert Taylor, of America, spoke of a serum made from the gonococcus, not a true vaccine.

Dr. MORGAN RICHARDS asked what was Mr. Baldwin's experience with graduated bougies in the chronic stage of gonorrhœa. He had found Fleck's bougies of very great use in the condition.

Dr. BUCHANAN said Mr. Baldwin mentioned that in certain cases he irrigated the posterior urethra and the bladder, and he asked by what method that was done.

Mr. REDMOND ROCHE said he recently saw gonorrhœa occurring in a boy, æt. 7 years. Such cases of acute gonorrhœa were comparatively rare, and, therefore, he thought a statement about it might be of interest. It was not clear how the child contracted the malady. There was a young servant in the house under suspicion, and though there was no history of immediate sexual contact, it was very likely that, owing to want of proper cleanliness, the child contracted it from the servant. However, the child had acute gonorrhœa with acute paraphimosis. He was called to see the patient in that condition with the view of adopting surgical measures to relieve the paraphimosis, and he did a complete circumcision. It illustrated the point made by the Chairman in advocating in as general a way as possible circumcision in the earliest years. If that boy had been circumcised—he had a particularly long prepuce—he would have stood a chance of escaping the disease. He had gonorrhœa in a very acute form, and it ran the usual course. He heard from the doctor the previous day that after seven weeks of alkaline and salol treatment he was now completely well. It seemed to him that the ordinary members of the Society would still be in a great deal of doubt as to what the line of treatment for active gonorrhœa should be in cases which resisted the ordinary simple measures. On the one hand they had heard from Mr. Pardoe words of admonition as to the danger of rushing in with active measures for fear of setting up conditions which were worse than the original state of things for which the treatment was adopted. The other side of the picture was presented by Mr. Baldwin, who advocated in a very hopeful way that active line of treatment in the great majority of cases which did not yield readily to expectant or milder measures of treatment. It would be a great pity if practitioners were deterred from using energetic measures which were likely to lead to good results in cases which were resistant to medical treatment, by reason of the fear of a more dangerous condition being set up. He thought that line of argument would be valid against almost any justifiable operative treatment for most ordinary diseases, even such a simple one as extraction of a tooth, because of the possibility that it might be followed by severe septic conditions. Therefore, he thought, practitioners should not allow that fear to unduly deter them from using strong methods in cases which seemed to need them. On the other hand, the active measures which were known to be necessary required more skill than the average practitioner, who did not deal frequently with such cases, could command, and he thought much care and discretion should be exercised in using such measures until they had ample opportunity of seeing them employed, if not employing them themselves. Reference had been made to trying to prevent gonorrhœa by legislative measures or by circumcision; he also thought it should be impressed upon patients when they had contracted the disease the necessity for treating it with great care and attention, more so than patients usually thought necessary. The laity seemed to have the

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idea which the medical student of old had, who, when asked how he treated gonorrhœa, replied: "I treat it with the greatest contempt." Certainly many members of the laity treated it as a very trivial incident, thinking that a few bottles of medicine procured at the chemists was all that the case called for. And when such patients consulted a doctor, the doctor did not always attach the significance to the disease which he should.

Dr. C. J. HARRISON said that he confessed that in the few cases of gonorrhœa which he had been called upon to treat he used the expectant treatment, and in the majority of cases he had done nothing more. His results had been very good. If possible he got the man to go to bed, and gave him bland things to drink, especially barley-water, and he also gave him aperients, alkalies, and hyoscyamus in big doses. As a rule he thought cases got well without injections. He had not had any experience of vaccine treatment, but from what it had accomplished in other septic conditions he thought it should prove to be very useful.

A MEMBER said he also could mention a very early case of gonorrhœa in a small boy, æt. 9 years. When called in to see him he found him obviously suffering from anterior urethritis of gonorrheal origin. The cause of his infection proved to be the servant of the house. The reason given for inciting the boy to sexual connection showed the great need for the education of the public on the matter. In Ireland certain members of the lower classes had the idea that the only genuine cure for gonorrhœa was sexual connection with some person who had never had it. In that case the servant went for the person in the house she was most certain of in that respect, namely the small boy. The case was treated by the method of active abortion, by irrigation. Protargol was used, and the recovery was rapid.

Mr. CAMPBELL BOYD said the practitioner in London seemed to get only rare cases of gonorrhœa to treat, though in the country many patients were seen. He had never had any trouble with such cases when using ordinary alkalies and sedatives and gentle astringents. Cases of gonorrhœa in London seemed to go to men in the City if they were in a good position, while others resorted to the chemist, who probably treated them. If the case was a bad one it went to the hospitals. Those circumstances combined to make the cases somewhat rare in general practice.

Mr. PARDOE, in reply, said they did not give the Germans priority or singularity in abortive methods of treatment; they were largely used in this country and were not confined to civil practice, for *sub rosa* they were used by many active and patriotic members of the profession in the Services. They could not be used as compulsory measures, because public opinion would not permit of that. He had rather hesitated to mention the German Imperial Navy in the present state of public opinion, but he had to do so in his opening because the statistics of that country were the only ones available of which he knew. He would not give any statistics of his own. With regard to the use of the syringe, he did not intend to imply that the ordinary urethral syringe was to be thrown into the dust-bin, but merely that in the acute stage of anterior urethritis the syringe had certain dangers when used by the patient himself. Certainly in the later stages of urethritis, which he had not touched upon, the syringe was most valuable. In acute anterior or posterior urethritis he did not like the use of the syringe. In answer to Dr. Parkinson's remarks about rest, he did not think that gentleman appreciated the differences which he (Mr. Pardoe) tried to indicate between the ambulant treatment of acute gonorrhœa and the resting treatment. What he tried to point out was that by the irrigation and abortive treatment patients were able to get about in great comfort and with practically no symptoms but by the simple expectant treatment, even when combined with rest, for some time patients had a horrible outflow of pus, but, of course, if the rest treatment could be combined with the abortive treatment so much the better. With regard to vaccines, it must never be forgotten that in the vast majority of cases gonorrhœa was not a pure infection, but a mixed one, and it would be found at all events after the first acute outburst of gonococci had subsided in the pus, that there were present such organisms as ordinary staphylococci *Micrococcus albus*, and not uncommonly other organisms. With regard to vaccines, obviously

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the one to employ must be the one which would attack with success the particular organism causing the disease. So far from agreeing that vaccines and inoculations with sera in acute gonorrhœa were disastrous, his experience was the reverse of that. He would like to hear what disastrous effects the last speaker had seen from the use of vaccines and sera in the treatment of acute gonorrhœa. He (Mr. Pardoe) had said in his opening remarks that he would not enter into details, but he would now mention a case of gonorrhœa. Not very long ago he was called in consultation to a young man, æt. 23 years, who was lying in bed with furious urethral discharge and all the symptoms of acute posterior urethritis and acute gonorrhœal cystitis. Gonococci were swarming in the pus, and they were recovered in large numbers from his urine in almost pure culture. He was bedridden because he had arthritis in both knees, both ankles, both elbows, and double purulent gonorrhœal ophthalmia. He was treated by a gonococcal vaccine, and eight weeks from the commencement of that most furious and virulent gonorrhœa the patient was able to play golf and to take all ordinary exercise. That was a good instance of successful treatment of acute gonorrhœa by vaccine. With regard to the chronic cases, very often the urethritis went on for months and even years, and although one could not recover the gonococcus in smears or in cultures from the centrifuged urine, one could recover a large quantity of the pus-producing micro-organism, and the vaccine used for that was one made, if possible, from a culture of the patient's own micro-organism. In answer to Dr. Mansell Moullin's question about anti-streptococcic serum, the serum which he (Mr. Pardoe) spoke about was the ordinary commercial Burroughs and Wellcome polyvalent anti-streptococcic serum, used in combination with the other abortive measures. The gonococcus would not grow in the presence of that serum. Mr. Roche had reproached him somewhat about acute and chronic gonorrhœa, but he (Mr. Pardoe) had not been speaking of chronic gonorrhœa at all. With regard to prophylaxis, he took it that the Chairman's objection to using prophylaxis was based upon personal antipathy, owing to the unpleasantness of the method. But in the German Imperial Navy a penny-in-the-slot machine was introduced on board the ships, and by the payment of two pfennigs, which was compulsory upon all liberty men, they got a box containing a little tube of protargol solution, or protargol in paraffin jelly, which they were instructed to use for themselves. The statistics which he mentioned were gained by the personal use by the surgeon of this method upon the men, but the men could employ the method upon themselves perfectly well. He had a word of concurrence to utter in regard to the importance of early treatment for the disease. The treatment of the disease would be far more satisfactory if patients would come for advice in the earliest possible stages. Even the simplest and quietest treatment by the expectant method, if instituted early, was far better than the most active measures if instituted late.

Mr. BALDWIN, in reply, said that in the treatment of the condition he had used the largest steel bougies which the urethra would admit, and it was useful to leave one in for some minutes before irrigations or installations were introduced. The pressure exerted by steel bougies on the walls of the urethra caused emptying of glands, absorption of inflammatory infiltration, and broke down and caused the destruction of granulations. With regard to the irrigation of the posterior urethra and the bladder, the same apparatus and method were used as in irrigating the anterior urethra—an irrigator and tube with a nozzle were used. For irrigating the anterior urethra the irrigator can would be about two feet above the patient's penis, but in posterior urethral irrigation the can should be raised three to four, or even, if necessary, six feet. The contraction of the compressor urethræ muscle had to be overcome, and that could be accomplished by getting the patient to strain down as if he were trying to pass water. That would often allow the fluid to pass into the bladder. The patient should be directed also to take a deep breath at the same time, and then, by raising the irrigator can sufficiently high, in most cases the fluid would go into the bladder. The patient would say when there was enough in the bladder, and it could be passed out. The bladder was filled twice a day and emptied out as often. If spasm of the compressor were so great that it would not allow the fluid to enter the bladder, or if the necessary pressure causes pain in the anterior urethra, a local anæsthetic can usually be "milked" back into the posterior urethra, when the trouble will be overcome. If not successful the practitioner should introduce the nozzle of the catheter attached to a Guyon syringe just within the anterior part of the compressor and put a local anæsthetic into the posterior urethra, and then introduce the nose of a soft catheter just within the grasp of the compressor, and irrigate through the catheter. As a rule, however, that was not necessary. If the directions as to the height of the irrigator and the method were carried out the bladder could be filled and the irrigation proceeded with without trouble.

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INTERNATIONAL CLINICS. Vols. I, II, III, and IV, 1907. J. B. LIPPINCOTT Co., Philadelphia and London, 1907.

IN these four volumes the practitioner will find a useful series of papers dealing with present-day problems in medicine and pathology, and with the newer methods of clinical diagnosis and treatment. The special lines on which the clinics are conducted have now been well established for some years, and evidently meet with the approval of the medical profession. Every branch of medicine and surgery is represented, and the contributors are men who are entitled to speak with authority on the subjects which they discuss.

The first volume contains as usual a summary of the progress of medicine during the previous year, but the subject is too large to allow of a useful description in the space allotted to it. An exception must be made, however, in the case of the surgical section, where Dr. Joseph C. Bloodgood has confined himself to two subjects—tumours and abdominal surgery. He has produced a very useful paper dealing with the advances in these branches of surgery, and has added a number of good illustrations, plain and coloured. On the whole the plain ones seem preferable to the coloured. Dr. Warthin writes on enlargement of the thymus, and tells all that is known about this mysterious organ. He concludes that "the best explanation we are able to give of thymic hypertrophy is that it is a compensatory hypertrophy of the gland, dependent upon a lymphotoxic or a myelotoxic exhaustion of the spleen, lymph nodes or bone marrow." Such an explanation cannot be said to clarify our pathological ideas on the subject very much. Professor Tuffier discusses the advantages of being able to operate within the pleural cavity without the induction of pneumo-thorax, and describes the various forms of apparatus which have been employed to secure this object. A large amount of experimental work has been done both in France and in Germany, and a chamber has now been devised in which the atmospheric pressure is so regulated that the lung does not collapse when the pleura is opened.

In Volume II Dr. Bertram Abrahams discusses the treatment of rheumatism. He points out the extreme care which must be taken in the case of young patients after one definite attack of rheumatic fever. Until the rheumatic age is over, that is, until they have passed their twentieth year, these patients should not be exposed to the strain of ordinary life, but should be guarded in every way. Of chronic rheumatism he says: "We are bound to confess that chronic rheumatism is a disease we do not meet with in practice, a disease which is virtually non-existent." This is a hard doctrine, and although we may whisper it confidentially to ourselves, and believe it, the public will still insist on the survival of the familiar diagnosis, which has brought consolation to so many. In an article on gonorrhœa and syphilis in infancy and early childhood, Dr. Morgan Rotch points out that it is the dissemination of a precise knowledge of gonorrhœa and syphilis which will best enable us to combat the ravages of these diseases. Definite knowledge of their manifestations and vivid portrayals of their direful results, purposely made hideous, should be brought before the laity. He is inclined to recommend a public crusade against these diseases, such as has been carried out in the case of tuberculosis, and anticipates an equally beneficial result. There are, however, obvious objections to any such form of publicity. He describes graphically the dangers of gonorrhœa in infants, and the risk there is of the infection being conveyed to other infants, nurses, etc., in any public institution for young children.

The opening paper in Volume III by Dr. Edsall on practical and theoretical considerations regarding dietetics—although the contents page gives it as "diabetes"—strikes us as particularly helpful, treating as it does of some guiding principles for the practitioner on this debatable subject. He refers to the difficulty the beginner has in obtaining a grasp of any principles at all, and points out that the teaching of physiology on the subject of diet must be tested carefully by the light of clinical experience. His own guiding principles may be summed up as follows: To determine accurately what is right and what is wrong in the patient's diet as arranged by himself; to begin, as a rule, with a rest and with simplicity of diet, but not to carry restrictions so far as to weaken the patient, even with the amelioration of certain symptoms; to meet difficulties, not by a mere reduction of the total amount of food or of a special form of food, but by substituting for the troublesome members of a class of foods others that yield a different digestive or metabolic residue, or which may be so prepared as to be more suitable; and finally, to determine not only about the foods but also what may be altered in the habits of eating, and in the use of fluids and food accessories. The practical application of these principles he describes with great clearness. Professor Robin, of Paris, believes that by the use of metallic ferments he has improved his statistics of lobar pneumonia. He admits that there is no specific treatment known for this disease, but thinks the metallic ferments aid the natural resisting powers of the system in overcoming the pneumococcal virus. The contents of Volume IV are of equal interest and value.

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## A LECTURE

ON

# DIET AND DIETETICS.


By FRED. J. SMITH, M.D., F.R.C.P.,
Physician to the London Hospital.


GENTLEMEN,—Diet and dietetics naturally divide themselves into three divisions, which I may thus tabulate:

In health of armies, prisons, etc.

In health of ordinary individuals.

In disease { special, *e.g.* diabetes, gout, etc. / general.

The first great division is an extremely important one, and very interesting to those who are responsible medical advisers to the authorities in charge of the establishments, but has not a great deal of interest to those whose chief duty is treatment of the sick.

The second is dearly loved by the faddists and the general public, to whom I will leave it, believing with many authorities that the ordinary instincts of the race are in the main reliable, though doubtless experience has taught many persons that they must regard themselves as exceptions to the general rules—exceptions which I must admit are very important in disease, as we shall see later on.

The third is a subject on which you can get very satisfactory information from your text-books, for it is often the most essential part of the treatment, and I do not propose to touch further on it.

The fourth to my mind is a peculiarly suitable subject for a clinical lecture, for in your text-books you will only find reference to a "light and nutritious diet" (so dearly loved by the candidate at an examination), or to adjectives such as "unstimulating," or "lowering," or "liquid," or "solid," applied to the diet recommended for any disease; and in your ward work the diets are ordered in a somewhat routine manner so that your attention is

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not drawn to details, and yet it is these very details upon which you will be expected to act as guides, not only in the first case you treat in private, but in practically ninety-nine cases out of every hundred throughout your practice.

To commence, let me try to impress on your minds one or two preliminary principles. The first is that in health we all habitually take considerably over the minimum required to keep us alive (Professor Chittenden has recently proved this to be even more true than we previously believed), and we all have within our bodies a considerable store of materials which can be drawn upon if necessary during a period of starvation, a period which Succi and others have proved can be very considerably prolonged provided great physical exertion be avoided. My generalised deduction from this principle as well as from other more commonplace considerations is that we shall do no harm to any patient by deprivation of food continued for a period to be measured more by his desires than by our preconceived notions.

The second principle to which I would specially draw your attention is that the function of the stomach of digesting food is one of health, and that in practically every disease, at least of an acute nature, it is interfered with, and in a very large number of diseases is for a time almost in abeyance.

The third principle concerns the appetite. Apart from what is known as bulimia or inordinate appetite, and a few cases of perverted appetite in neurotic or hysterical patients, the appetite is a function (used in a mathematical sense) of the health and digestive powers of the stomach, and does actually serve in some degree as an outward index or indication that the stomach is fit to receive and to deal with a supply of food, and my main contention is that it may be used as such an index.

In addition to this general use of the word "appetite" as meaning a desire for food, let me beg of you not to lose sight of its more special meaning as an inclination or desire for some *special kind of food*, and let me lay down this rule for you: do not forbid a patient an article of diet which he thinks he can enjoy, simply on account of some preconceived notion of your own that custom has not sanctioned its use for an invalid. Let him have a small taste of it and try, provided he tells you that when well he can enjoy and digest the given article; previous experience on the patient's part is worth a very great deal of theory on your part, and he may, as a general rule, be safely trusted with a very small quantity of the article, provided it be not obviously and not intrinsically unsuitable, such, I mean, as a raw turnip, or potato, or other hard fruit or uncooked vegetable. I do not wish to beg the whole question by using the terms "obviously" and "intrinsically," and my illustrations are nearly the only ones I can think of, for in view of the extreme variability of the human stomach for digesting or refusing to digest in health such things as pork, veal, bacon, ham (cold boiled ham is one of the most easily digested of foods), lobster, fish, milk, fruit, etc., I think that a preconceived theory may do untold mischief by depriving a convalescent of the only thing he can fancy, while the ascertained fact may bring comfort to his mind and rouse in him once more the desire of life, if it be only to satisfy the pleasures of the palate.

Laboratory experiments may help us here a little in pre-determining the digestibility of a given article, but do not put too much trust in them: they are not the same thing as the palate and the living stomach working in harmony, and assisted by the teeth or by artificial means of comminution of the food to be taken.

Taking these principles in the gross as a complete chain of argument, they say at once—do not feed sick people too much, and let their appetites, general and special, be in great measure the guiding rule in feeding them. If I were to stop there and say nothing more, I should have enunciated a principle which, even if abused, would lead to very little harm, and if followed with reason would, I am sure, give sick people an incalculable amount of relief from discomfort and thereby promote their recovery, when recovery is possible, and make the passage to the grave a reasonably easy one when recovery is impossible. But, obviously, I must enter into some little detail for you if my lecture is to be of any use.

Let us first consider a few of the articles of dietary: milk, eggs and sugar, meat, and later a few diseases in which they may be used.

A word or two on milk: It is common knowledge that for seven or nine months a child should have nothing but milk; hence arises a superstition

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that milk is the most easily digested article of consumption under all and any circumstances—a superstition to which I beg to notify to you all that I do not blindly subscribe, but feel compelled on the contrary to raise my voice as strongly as possible in opposition. It would, on the other hand, be equally untrue to say that it was the most indigestible article of diet in the world, though my experience in the post-mortem room and by the bed-side would lend a good deal of colour to the statement, which I firmly believe comes nearer to the truth so far as sick people are concerned than the original idea.

[I am paying, of course, in this lecture, no regard whatever to milk as a carrier of infection—tubercle, scarlet fever, typhoid, etc.—but simply concerned with it as an article of diet for those acutely ill, and am presuming it free from extraneous matters.]

First as to quantity and concentration: In most cases of acute illness, especially pyrexial, thirst is rather a prominent feature, and I wish to warn you against a routine practice of leaving a jug of milk and a glass within reach of a patient to help himself. Milk, to the palate, is only water, or a thirst quencher, but to the stomach is very much more, and when taken by the glass-full in its natural state is very prone to curdle in the stomach into a large, solid, and hardish mass, with which the organ may have a good deal of trouble in dealing, though this need not necessarily be the case, and you may find a patient desirous of having milk constantly at hand, and find, too, that he has no trouble in disposing of it. I believe this class to be the exception, and I would lay down the following guidance for you: put only the syphon of soda or the water-jug near the patient. I may say *en passant* that water which is *fit for a healthy person* to drink is fit for one who is acutely ill, and that boiling the water is not only unnecessary, but makes it much less palatable, though I have often had it suggested to me that water should be boiled and cooled before a sick person should drink it. I cannot imagine whence arose the idea that water in which toast has been soaked is better for a patient than plain water; it may be that the source of the water was originally suspicious, hence the water was boiled and found so unpleasant that the toast was added to give it a flavour—to some people most unpleasant, for I well remember to this day my own sufferings (it was nothing less) from being only allowed toast-water during an attack of measles. I suppose, too, there is a small amount of sugar in such toast-water.

I say put only the diluent within reach of the patient and let the nurse add milk in quantity never to exceed two ounces to the drink, and generally much less, according to the wishes of the patient and the nature of the illness. Of the latter variant I would give as illustration all forms of gastritis or of direct vomiting, *e.g.* from heart disease affecting the gastric circulation; in such cases as these the symptoms are our best guide; if plain water causes vomiting the supply must be placed out of the patient's reach and given into the nurse's hands, and the milk must be entirely suppressed for a day or so and resumed in quantities not greater than a teaspoonful.

It is in such cases as these I have learnt by experience that milk, as a rule, is a by no means light and nutritious diet; light it may be, but nutritious, no, for it only causes vomiting, and I am in the habit of ordering a dry biscuit or bread crust which *must* be well masticated before it can be swallowed, or even a tiny bit of meat, but often enough absolute starvation is the only means of relieving the extreme irritability of the stomach.

So much for milk in its pure state, but there are many other what we may call compounds of milk that are available: for instance, the simplest is curds and whey, or junket, which means that the milk is artificially curdled by a little rennet before use and the resultant curd can be dealt with in any way desirable, *e.g.* flavoured with sugar and merely masticated with the tongue or again broken up with forks or other implements. By suitable contrivances this can be done so finely that when mixed with a liquid the resultant hardly differs in physical qualities from milk, as witness Hutchison's diabetic milk, which is prepared in this manner so far as curdling is concerned. By this means alone I think a very suitable food for sick young babies can be easily prepared at a minimum of cost and trouble, and is well worth a trial.

Then, again, we have the liquid custard, which we look upon, when nicely flavoured, as a most suitable material for an invalid. It contains the yolks (only) of eggs in addition to the milk; three eggs are an approximate quantity for a pint of milk and flavour to suit, the important point being that in the making it is not to be boiled, but only warmed

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or heated nearly to the boiling point. Very similar in composition is the baked custard; it differs from the liquid in that it is practically raised to boiling point in the oven; at any rate it is a solid and requires to be broken up with the tongue.

Next in order of complication come arrowroot and cornflour and hasty pudding, small quantities of which may be given *at the wish of the patient*, and still more complication with milk puddings of any sort; it is the wish of the patient that I here wish to emphasise, for some patients do not like this kind of food.

Again, we have soups in all variety, not one of which can, if the patient fancies it, do much harm provided they are not made with commercial articles of diet. All these I would absolutely banish from the sick room wherever possible, and what I mean by "possible" is if there is a kitchen in the house of the sick, and some person—cook or another—who can follow mere simple directions, and these elements exist in practically all private patients' houses and even in the houses of the poor. I know, of course, that these things are used literally by the ton in cookery, both for the healthy and the sick, and they have their uses as flavours, and occasionally, very rarely, as stimulants for a crisis, but they are precious little use as foods or nourishers of the system. I may now say a word or two on meat, and I will begin with a simple receipt for beef tea, which is this: Cut up a half pound of lean meat (I do not think it matters what) into small knobs about the size of ordinary dice, and place in a small dish with a proportion of one pound to the pint of water and a drachm of vinegar (dilute HCl will do) and gr. xxx of salt and let it stand for an hour, or even two; then place in a slow oven and bring it up to just below boiling point. Practically let it stew for an hour, and then pour out into a vessel and feed the patient on the fluid or on the more solid remnants, which will not be bulky, for the acid salt water will have dissolved practically the whole of the lean meat, but will not have broken it down into simple extractives. Even this may not suit the patient's tastes. And now I have another word of practical wisdom for you: do not refuse permission to have it flavoured with sauces if the patient prefers it and asks for it. They cannot conceivably hurt a patient, and yet I never find them allowed—nobody could ever tell me why.

Now a word or two on eggs: A complete egg beaten up in milk is very nourishing, but may be distasteful; they may be boiled or poached, or even buttered, but again let me add that it is the taste and appetite of the patient that has to be considered.

Of actual objects to be bought at a butcher's, tripe is the most digestible, and *if the patient likes it* is suitable for any patient. Sweetbread is good too, but it is said to be very rich in purin-bodies (the latest bugbear of the dietician), no doubt bad things for the gouty man, but they need hardly trouble you for the acutely ill: *small* pieces of beef and mutton finely cut cannot hurt anybody, and their use is a mere question of appetite.

The value of fish lies in the shortness of fibre of their muscles, and the consequent ease with which portions can be broken up into small fragments, and I rather fancy that the same argument holds with regard to bird of any sort as an article of diet, for it is, I believe, a curious fact that in a laboratory experiment similar-shaped and sized pieces of chicken digest more slowly than the corresponding pieces of beef and mutton, and hence I can only believe that the palate has more to do with the universal habit of letting convalescents have a bit of chicken than has the digestibility of the meat itself.

*Artificial digestion.*—A few words on artificially digested foods may not be out of place: In such cases as acute pneumonia, severe smallpox, etc., you may often be tempted into trying to give predigested food to a patient, for it may happen that real exhaustion from want of nutriment may supervene. Personally, I think such cases are rare, and more frequently exist in the anxiety of doctor and friends than in the reality of actual starvation. When physiologists speak of the absorption of nitrogenous matters they always use the plural in speaking of peptones, believing that there are many products of nitrogenous digestion differing in slight molecular points from one another, and quite possibly having slightly different uses for different individuals. If, now, we take some peptonising or other digestive powder, and with it carry on *in vitro* the digestion of some food and give this to the patient, I have the following objections: first, that such digested materials have commonly a very unpleasant taste; and secondly, that we can never be quite sure that

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we have carried the process just to the right point and not beyond, so that we may, after all, not be helping but hindering the stomach and tissues of our patients. What I think is a much more sensible and practical plan is to administer the digestive powder to the patient, give it, say, ten minutes' start, and then give the food; by this means we avoid the nasty taste, and we also give the stomach the chance of doing what it can on its own with the food, and also of finding and using what it can out of the digestive powder, and so taking control, as far as its state will allow, of the digestive processes which the powder may initiate. There may not be much of value in this proceeding, but it seems to me much more rational than to think that artificial digestion can take the place of the natural act.

*Times of feeding.*—One's natural times of feeding in health may be said to correspond roughly to about every four hours during an ordinary period out of bed, say roughly, 8, 12, 4, 8, ; and this we have already admitted leads to our accumulating a store of nutritious material, in fact, to be an excess over our absolute requirements. We have also seen that in acute illness the capabilities of the stomach for converting food into available nutriment for the tissues is considerably diminished, and yet there seems to me to be a desire on the part of those nursing a case of illness to feed the patient at intervals of two hours or so. The reason for this desire seems to me to be even less defensible than that for not allowing the patient the kind of food that he wishes. One can possibly understand in the latter case the fear of inducing indigestion, unjustified though I believe even this fear to be, but I cannot understand the reason for repeatedly loading up an organ with work, the capacity of which for work is admittedly below par. It may be argued that the amount of food at each meal is small—this I admit; and further, if he is not fed frequently he will fail from exhaustion—this is the point upon which I am very sceptical. I have never yet seen a patient die in, say, the first ten days (this may fairly be taken to indicate an acute illness of which we are speaking) of an illness from deprivation of food, but I have seen plenty in whom vomiting has been induced by attempts at feeding too frequently.

Another consideration crops up here: I say without fear of contradiction that the very best medicine outside or inside the pharmacopœia is natural sleep, and what chance is there of getting a good dose of that if he is to be wakened at frequent intervals to take some food which as likely as not he loathes.

No, gentlemen; let me again beg you to give the patient a chance, and if he objects to two-hourly feeds to let him have his way and never wake a patient out of sleep simply to feed him, except in some very exceptional cases of prolonged exhausting illnesses, which we are hardly considering now, though almost all I have said might be taken as applying to such cases as phthisis, recovery from typhoid, etc.

Let me now take one or two illustrative diseases.

*Morbus cordis.*—In a large number of these cases there seems very easily to be established a vicious circle; the stomach is congested by back-pressure and refuses its office; this leads to vomiting and to palpitation and cardiac distress, and also exhaustion. So much is this the case that in a patient's own diagnosis the stomach is blamed for the heart's misdeeds, and the heart often enough for the stomach's misdeeds. Now *qua* feeding and sleep, I am very strongly convinced that the best way to break this circle is to begin with absolute starvation and a small dose of morphia, and when sleep is procured to let it last as long as it will (it is not quite germane to my subject, but I cannot refrain from once more insisting upon the fact that morphia in small doses is *not* a cardiac depressant, but either directly or indirectly through the sleep is one of the most valuable cardiac tonics we possess when used with common sense). Let the sleep last, I say, as long as it will, and then when the patient wakes give him a *very* small quantity of food if he asks for it, and begin by asking what he would like. I have very often found that in such cases the stomach will tolerate and use a small quantity of a dry or solid diet, such as a biscuit, a crust of bread, a tiny piece of flesh of some sort, when it will not tolerate a draught of milk or milk and soda. I have no objection to your trying small quantities, say dessert-spoonful doses, of milk if the patient likes it, asks for it, and does not vomit after the first trial, but do not refuse him half an ounce of meat if he asks for it simply on the ground that you fear he will not digest it. Again, if he is obviously dying of cardiac failure, let me beseech you not to embitter his last moments by insisting on his taking food, thus very likely blowing him up

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with wind if it does not actually cause a fatal effort of vomiting, so effectually preventing any possible chance of recovery that he might otherwise have had. You have only to see one or two such cases and watch how their one concern is to be able to breathe to feel the truth of my assertion that loading the stomach with large quantities of fluid will not help the breath.

*Acute nephritis.*—On the dietetic treatment of nephritis many views are held, but all would agree that during the very early stage it is well to withhold food of almost any description, and my own practice is to entirely withhold it for twenty-four or forty-eight hours; and I must admit that subsequently the best diet is in all probability milk for some week or ten days, or at least one or more of the many forms of milk or milk with carbohydrate material in the shape of rice, ground rice, sago, tapioca, arrowroot, cornflour, etc. But let me advise you not to keep this on too long, and by too long I mean beyond the limit of the patient's taste for it; if in, say, three weeks or so he begins to ask for a more substantial diet of ordinary food let him have it—I do not think you will be doing the kidneys any harm. It is usually alleged that the nitrogenous material in the food causes either by the so-called luxus consumption, or by more ordinary channels an excess of work for the kidneys. I believe that even the present-day scientist has given up this view, and I am sure that if you will look at the matter from a broad, practical point of view, you will be puzzled to see how starving the rest of the body can possibly assist in restoring health to the kidney cells. It is quite proper and legitimate to give damaged cells as little work to do as possible, but this is a very different matter to trying by a milk diet to give them no work at all—a feat which is impossible while life is still being carried on in the rest of the body. It is a little wide of our present mark, and yet not altogether foreign to it, to mention to you that the prognosis and progress of a kidney case is to be estimated by the feelings of the patient, and by the degree and progress of the anæmia and flabbiness of the tissues rather than by the amount of albumen in the urine; and the deduction from this is very much to the point in our present view, viz. if you are relaxing the diet of a nephritic, consider the anæmia and the general condition of the patient as expressed by himself rather than estimate the amount of albumen in the urine, which, after all, is more a measure of some leakage through, rather than present health of, the kidneys as a whole.

*Primary intestinal troubles.*—It is not easy to lay down any very rigid rules in this class of case, for every rule will have many personal exceptions, but I think I can for all that give you one or two to which exception can hardly be taken.

First I would say, do not be afraid of death from starvation from the diet point of view; if the stomach is so profoundly irritable that vomiting is caused by any and every thing put into it, death may possibly follow from that cause, and you must try rectal or some other method of feeding and nutrition. I would, however, say that death from such a cause must be very rare, and I should say, could not possibly occur under a week unless accelerated by exhaustion caused by the act of vomiting induced by injudicious efforts at feeding. I have no very great belief in the efficacy of rectal feeding, so far, that is, as the absorption of material from the rectum is sufficient or even appropriate for the needs of the body, but for all that I permit it to my house-physician, and do not dissuade a practitioner who consults me from trying it, and this for two or three reasons. First and foremost it permits the medical man who does it to think that he is doing good—a permission and belief that is by no means to be despised in these days of scepticism. Secondly, more important, it certainly can be made to keep within bounds the thirst of the patient, which is apt to be very worrying during this period, and it also has a considerable moral effect upon the patient whose mind is thus set at rest as to real starvation (this is a by no means unimportant effect upon the minds of the friends too). Thirdly, and I certainly think most importantly, it gives time to the stomach to recover some degree of control by putting it at absolute rest, time which the natural fears of doctor, patient, and friends, will very frequently not allow.

I shall not trouble you with the details of a nutrient enema; you will find them in our pharmacopœia, but I would give you this caution: the administration of an enema is a disturbance to the patient, and should therefore be carried out at a time just preceding that natural to rest, so as to serve the double object of not breaking into this period of rest, and also of adding to the chances

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of retention and absorption of the nutrient part of the enema. Apart, however, from nutrient enemata, I would advise you not to be bound by tradition in finding and trying any food substance for a sick stomach. I have seen many cases in which I have been met with the remark: "The patient cannot keep anything down; the smallest quantity of milk causes vomiting." It is amazing how traditional teaching seems to have simply killed any common-sense originality in this particular direction, and to hear the above cry repeated so often one would be tempted to believe that milk was the only food substance the world could produce. It is in precisely such cases I have scored my few real triumphs, for after the first shock of incredulity and belief in my insanity has subsided, the doctor and friends have allowed reason to overcome prejudice with brilliant results.

Only the other day, to take an example, I saw a boy, æt. 9 years, who seemed at intervals to get attacks of vomiting for which no reason could be found, and I must say I was somewhat frightened about the onset of tubercular meningitis when his latest attack had lasted two or three days beyond the average. However, I asked him what he would like instead of the slops which he had been having *ad nauseam*, and on his replying something solid, I ordered a mutton chop to be bought and the boy to be given about half an ounce of the tenderest part with a tiny piece of bread: he ate it and was not sick again. You may take this as an illustration of my meaning. Or take again a case of gastric ulcer which has been in bed for a week on bread and milk, arrowroot, baked apple, etc. The Germans are in the habit of allowing such patients (I am told by one who has been there) a small piece of boiled ham as being the substance most easily digested, provided the patient likes it. If you cross-examine such patients you will be astonished at the number who will confess that they get just as much pain from milk or slops as they do from a small piece of meat—a further proof if any were needed that milk is no improvement upon other articles properly divided up.

*April 20th*, 1908.

# HARMLESS ALBUMINURIAS.*

By LEONARD WILLIAMS, M.D., M.R.C.P.
Physician to the French Hospital;
Assistant Physician to the Metropolitan Hospital.

For some months past I have been increasingly impressed with the very restricted view which is commonly taken of the significance of albuminuria. I have, therefore, thought that it might be not unprofitable if I endeavoured to present this symptom to you as something which, in itself, is perfectly inoffensive, by which, nevertheless, the unwary allow themselves to be frightened into a condition where judgment fails and perspective no longer exists. Such a state of mind on the part of the physician is liable to be a very serious matter to the patient, who, merely because some albumen has unfortunately been discovered in his urine, may be refused for life insurance and subjected to a line of treatment which is diametrically opposed to that of which he stands in need, with the result that he is not infrequently transformed from a robust and useful member of society into a whining and useless hypochondriac.

It is not that I claim any originality for this attitude. Physicians of great eminence and distinction have, for some time past, been protesting against the view of the matter which I am here to deplore. As long ago as 1900 Saundby,† of Birmingham, exposed the fallacies upon which the accepted view was based. Last year Rose Bradford‡ did the same, and in the interval Sir Almroth Wright,§ W. Collier, of Oxford, and many others, both in this country and in France, notably Dr. Teissier,‖ of Lyons, have preached convincingly on the same text. That the sermons of these authorities do not appear to have been sufficiently heeded must be my excuse for taking up the parable.

The most practical method of dealing with the matter would seem to be to enumerate some of the conditions in which albuminuria is liable to occur and to attempt to bring them under some kind of

* A lecture delivered at the Medical Graduates' College.
† 'Lectures on Renal Diseases' (Wright, 1900).
‡ 'Brit. Med. Journ.,' March 30th, 1907.
§ 'Lancet,' October 21st, 1905.
‖ 'Les Albuminuries Curables' (J. D. Baillière).

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common denominator. Let us, then, first of all consider the physiological aspect of the matter.

We have it on the authority of Virchow, Martin, Hofmeister, and others that albuminuria is the rule with new-born babies. The amount of albumen is at first abundant, but diminishes gradually, until by the fourteenth day it has completely disappeared.

Adolescents, who are apparently in good health, are subject to what is described as cyclical or postural albuminuria, a phenomenon which is now admitted on all hands to be devoid of pathological significance. Amongst adults of mature age, military men and doctors, Leube and Furbringer have described a considerable number of cases of cyclical albuminuria, unaccompanied and unfollowed by any morbid tendency.

Adolescents, who are undoubtedly in good health, readily develop albuminuria as the result of fatigue. This has been conclusively shown by W. Collier, of Oxford,* who examined young men in training for races, men who may therefore be assumed to have been in a high state of physical and physiological efficiency. In a very large percentage (from 57 to 100) of these young men albumen appeared in the urine a short time after hard exercise, to disappear again after a period of repose.

According to Playfair,† albuminuria occurs in 20 per cent. of pregnant women after the third month, and in a much larger percentage of primipara. Some of these cases, it is true, progress to definite renal disease, but the vast majority suffer no inconvenience from the occurrence of the symptom.

Finally, there is Senator's statement to the effect that every urine will be found to contain albumen if sufficiently concentrated.

The foregoing are, all of them, admittedly physiological conditions, the significance of which it is necessary to explain if we would realise the meaning of albuminuria as it occurs in conditions which are not physiological. Now it is universally conceded that albuminuria may be caused by a congested state of the renal vessels—a fact with which we are all familiar in heart disease and similar conditions. Perhaps the most striking instance is afforded by the albuminuria due to injuries of the spinal cord, in which the vaso-motor nerves are paralysed. But lesser degrees of congestion will produce the same results. Chills to the surface, for instance, as after cold bathing, especially in the sea, are known to provoke the appearance of albumen in the urine, presumably by increasing the amount of blood in the splanchnic area, and thus inducing a transient renal stasis. Let us see what bearing this has upon the physiological albuminurias just mentioned.

The newly-born infant is but partially adapted to the new conditions to which he is suddenly subjected. Most of the powers which he subsequently develops are in a very embryonic state. More especially is this true of the co-ordinating or controlling power. He can, for instance, use muscles, but he has not learned to co-ordinate them; his sphincters act, but he is unable to control them. These powers are developed only by degrees and by training. We must, I think, assume the same to be the case in a much higher degree with the complicated mechanism of vaso-motor co-ordination and control. The blushing and pallor which occur involuntarily on any slight emotion, even at a very much later period, are sufficient to show that this mechanism is an exceedingly delicate one, which demands a considerable amount of training and adjustment before it can be considered to be in good working order. When a baby comes into the world he leaves a warm and equable climate for one which is cold and liable to considerable variations. However carefully he may be protected from such influences they are bound to reach him, to the extent, at any rate, of causing very decided differences in the balance of blood distribution to which he has hitherto been accustomed. His skin is now, for the first time, called upon to contract, and this contraction causes the blood to seek refuge in the organs of the splanchnic area. The vessels in this area have not yet learned how to behave in the presence of such an influx, and the organs suffer a certain degree of congestion. The renal vessels are among those affected, and an albuminuria results. After a few days, the splanchnic vessels learn their lesson, the congestion is gradually reduced, and the albuminuria disappears.

The case of the adolescent is not very different from that of the newborn baby. As the boy is being transformed into the man, and the girl into the woman, new activities are developed, which place

* 'Brit. Med. Journ.,' January 5th, 1907, p. 4.

† 'Handbook of Midwifery,' 2nd edition, vol. i, p. 222.

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an enormous strain upon the delicate vaso-motor mechanism, which tends, in consequence, to fail. Failure of this mechanism is almost invariably in one direction, in that, namely, of undue vaso-dilation. The calls upon the nervous system are so considerable and so pressing that the tone of the peripheral arteries is not adequately maintained, with the result which always ensues in such circumstances, namely, that during the erect posture the blood collects in the capacious vessels of the splanchnic area. Then ensues a congestion in the renal vessels with consequent albuminuria. So long as the patient is recumbent the blood does not tarry unduly in the splanchnic area; there is no renal congestion and no albuminuria. No sooner, however, is the erect posture assumed than the albumen reappears. The fact that in some of these cases the albumen diminishes or disappears towards the end of the day only signifies that by that time the vaso-motor mechanism has been provoked into a sense of its responsibities, and has realised the necessity for contracting the splanchnic vessels in response to the change of posture. This postural or cyclical albuminuria is quite common in people who have been obliged to keep their beds for a considerable time, for some such cause as a fractured thigh. In them the vaso-motor response atrophies from disuse, so to speak, and has to be re-educated as the erect posture is resumed.

As an instance of what is liable to happen to an adolescent who exhibits the phenomenon of postural albuminuria, let me briefly relate the case of a young man, æt. 23 years, whom I first saw last July. Eighteen months previously, after he had been working hard at the University for an examination, he tried to insure his life, but was refused. This naturally alarmed him, and he consulted his doctor. The latter found that his urine contained no less than 40 per cent. albumen, told him that he had Bright's disease, and ordered him to Cannes for the winter. I may say parenthetically that if the patient had really had nephritis, the Riviera with its abrupt variations of temperature was about as bad a place as could possibly have been selected for him. However, to the Riviera he went, and returned home no better. His doctor then sent him to bed and put him on a diet consisting only of milk. In about a week the albumen had completely disappeared, whereupon he was allowed up again. In two days the albumen had re-appeared, but in reduced quantities, and he was sent back to bed. Again the albumen disappeared, to re-appear as soon as he was allowed to get up. My notes do not say exactly how long this game of battledore and shuttlecock continued, but eventually the patient wearied of it. Having, in consultation with his friends, decided that appendicitis could be the only explanation of the mystery, he came up to town to consult a surgeon, through whose instrumentality he eventually came to me, still, curious to relate, having his appendix intact. He was also wearing three or four layers of thick woollen under-garments, his skin was relaxed and sodden, his blood-pressure was 85 mm. Hg., his urine scanty and loaded with albumen. The most careful examination failed, however, to reveal any evidence of organic disease, so, when I had satisfied myself as to the absence of albumen after a few days in bed, and the absence of granular casts at all times, I instructed him how to clothe himself. I sent him to Margate with some strychnine and calcium chloride and told him to pull himself together and live hard. Being anxious to get well he did as he was told, and is now working hard, taking plenty of exercise, and feeling perfectly well. He has learned how to test for albumen, and he tells me that, except after a dance or a hard set at tennis, his urine is now quite free from it.

The albuminuria of young athletes described by Collier, and previously by Dunhill,* of Melbourne, it not quite so easy of explanation as the foregoing. It is nevertheless safe to assert that it is purely vaso-motor in origin. The train of events is probably somewhat as follows: We know that during muscular exercise the general blood-pressure is raised. The heart increases the number of its systoles, the vessels in the splanchnic area are contracted, while those which supply the muscles actually in use are dilated. After the exercise is over there is a reaction, which varies in degree directly with the magnitude and duration of the effort. After severe and prolonged exertion, therefore, the splanchnics dilate, and the muscular vessels tend to contract. If this contraction is hastened, as it generally is in young men at universities and public schools, and as it was in all

* 'Intercol. Med. Journ. of Australasia,' July 20th, 1902.

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Dunhill's* cases, by a cold shower or plunge, the blood is driven inwards with such rapidity that a renal congestion is produced, and albuminuria results. It is probable, however, that apart altogether from the cold shower, the reaction from the conditions which necessarily obtain during active exercise are sufficient to induce a renal stasis, more especially in young men in whom we have seen the vaso-motor response to be but partially educated. It seems, however, that this may not be the whole explanation, for not only is the composition of the blood materially altered by vigorous exertion, in that the waste products are largely augmented, but the sudden and considerable elevation of blood-pressure entailed by such exertion is of itself sufficient to provoke a transient albuminuria.

This albuminuria of young athletes, whatever be its explanation, is a very important fact to remember. Failure to do so has on many occasions given rise to serious results. In one such instance which is known to most of us, the results, if not serious, are certainly prodigious. It is that of a gentleman who was formerly a scholar of King's College, Cambridge, and a lecturer at the university, who now runs a restaurant on special dietetic lines near Charing Cross. This gentleman was a very distinguished athlete, both at school and college, and won championships in tennis and racquets for several years in succession. When at the zenith of his fame his urine was examined and found to contain albumen. Thereupon this Honours Coach (for such he was) and athletic record-breaker was duly pronounced to be suffering from Bright's disease — an opinion which was confirmed by at least two other responsible medical men. Being quite unconvinced, the patient took to studying the question of diet and experimented on himself, with the result that, according to his own view he so managed matters that the albumen disappeared, and with it various other minor troubles to which he had been subject. The albuminuria in his case was evidently merely an athletic albuminuria, perfectly harmless and physiological, which would have disappeared either in the ordinary course of events or by the exhibition of calcium chloride or strychnine, or both. One consequence of attributing it to Bright's disease has been the erection of the very elaborate system of dietetics whose temple is to be found near Charing Cross. Mistakes in diagnosis are always unfortunate, but if each mistake is to be followed by a new dietetic panacea, the life of the medical man, to say nothing of the layman, will be beset by complications and conundrums insuperable. Let us then admit the possibility of athletic albuminuria, and do all we can not only to recognise it, but to place it in its proper category among harmless and even possibly beneficent manifestations.

The presence of albumen in the urine of pregnant women after the third month is obviously mechanical. The enlarging uterus, even if it does not exercise direct pressure upon the renal veins, materially alters the distribution of pressure in the splanchnic area, and if the normal compensatory mechanism is not in good working order, a stasis with consequent albuminuria is not difficult to imagine. It is a significant fact that such an albuminuria is more common in primiparæ.

We have, then, in albuminuria a symptom which may occur, and constantly does occur, in conditions which are purely physiological. We know, further, that it occurs in states which, though pathological in various degrees, are nevertheless not such as even to suggest that the kidneys are at fault. I have already mentioned cardiac disease and spinal injuries, but these by no means exhaust the conditions in which albumen is frequently, if not constantly, found in the urine. It is very commonly present in chlorosis, in Graves' disease, in tonsillitis, even other than diphtheritic, in some forms of dyspepsia, and in almost all cases of hepatic congestion; in migraine, in epilepsy, and in most toxic pyrexias. Among pulmonary conditions it is found as an early sign of tubercle; it is common in asthma and by no means uncommon in the bronchitis of emphysematous patients. Moreover, a very large number of drugs will give rise to it: cubebs, copaiba, turpentine, mercury, morphia, quinine, arsenic, and phosphorus are among the most important, but they by no means complete the list. Passing abnormalities anywhere in the urinary passages may provoke the symptom. An excess of oxalates in the urine will produce it; so will the irritation of small calculi or sand. A slight cystitis is by no means an uncommon cause, and an exceedingly common one is the presence of spermatozoa in the urethra. Altered

* 'Brit. Med. Journ.,' April 27th, 1907, p. 1031.

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blood states, as in the essential anæmias, almost always provoke albuminuria.

It seems scarcely credible that a symptom which is common to so many diverse conditions, both physiological and pathological, should have succeeded for so long in masquerading as necessarily connoting renal disease. And yet it is within the experience of all of us that people have been, and, alas, are still being, refused for life assurance and otherwise condemned as damaged individuals merely because, from some of the above-mentioned causes, a little albumen has been found in their urine. It would be just as logical—it would, indeed, be more reasonable—if dyspnœa were regarded as necessarily indicating pulmonary or cardiac disease. Dyspnœa is in many cases very significant of such disease, but inasmuch as we have all of us become very breathless hundreds of times in the course of our lives without any untoward effects, we have acquired some sense of perspective in the matter. It would be a good thing if the presence of albumen in the urine could be manifested by some sign equally gross and obtrusive. We should then come to realise how frequent an occurrence it is, and how seldom it indicates anything more serious than a passing change of pressure in the blood-vessels of the splanchnic area. Albuminuria, like dyspnœa, may be the expression of very grave and fatal diseases, but, like dyspnœa, it may also indicate nothing more serious than a considerable, though perfectly harmless, alteration in the distribution of blood-pressure. We all make it a rule to gauge the significance of dyspnœa by the causes which provoke it. No one would dream of shaking his head and crying "heart disease" because a man was breathless after a mile race; but it cannot be said that no one could be said to shake his head and cry "renal disease" because the same man produced albuminous urine after the same ordeal.

Very often, of course, the meaning of an albuminuria is sufficiently obvious. It is seldom, indeed, that we find ourselves in doubt about acute nephritis, or chronic tubal nephritis, or amyloid disease. In these cases and in gross tuberculous renal lesions, the co-existing symptoms are almost invariably such as to point unmistakably to the true source of the albumen. The cases which give rise to perplexity are those in which the albumen constitutes the only, or almost the only, symptom. In such cases one has no right to express anxiety, much less to pronounce a sentence of incurable disease, unless in addition to albumen the urine also contains definite evidence of structural disturbance in the kidneys. This ought to be an absolute rule which permits of no exceptions. Such evidence is afforded by the presence of epithelium, of blood, of tube casts, more especially of the granular or fatty variety. Hyaline casts, although they are suspicious, have not the same significance, for not only may they be found in small numbers in almost any urine if sought for with sufficient diligence, but they are often present in large numbers in nervous and febrile conditions which have no renal connection. It must be admitted, no doubt, that a specific gravity of less than 1015 is a suspicious factor, but inasmuch as nervous people very often have urine of low specific gravity, it affords no excuse for any relaxation of the above rule. I am prepared to go even further than this, and to dare to call in question the teaching of the text-books by saying that albuminuria, even when accompanied by a low specific gravity, by an enlarged left ventricle, and by a definitely supernormal blood-pressure, is no necessary indication of renal disease. For, granted the high blood-pressure, both the albuminuria and the enlarged left ventricle follow as a matter of course. It is fortunately no longer necessary to insist that high blood-pressure may be a primary event, in the sense that it may, and, indeed, generally does, occur quite independently of any structural disease in the kidneys or elsewhere. The teachings of Sir Clifford Allbutt on this subject have at last secured the attention which they have long deserved, and it is now admitted that in the vast majority of cases it is the structural disease which follows the high blood-pressure, and not, as used formerly to be believed, the high blood-pressure which follows the structural disease. Now, a person with this simple "hyperpiesis" as it is called, may complain of various symptoms, most of them so indefinite that he postpones consulting his doctor. When at length he does so, the latter will generally find a urine of low specific gravity containing a definite amount of albumen. Examination of his cardio-vascular system will probably reveal an enlarged left ventricle, and certainly a blood-pressure of about 200 mm. Hg. Now, I say again that one has no right to pronounce such a patient to be the subject of kidney disease, and treat him as such,

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unless, in addition to the albumen, the urine contains epithelial, or granular, or fatty casts—unless, that is, he shows unmistakable evidence that the albumen is due to structural alteration in the kidneys, and not to some functional disturbance of the vaso-motor mechanism. We have in the centrifuge and the microscope two instruments which are capable of settling this point quite definitely, and until their verdict is available, to assume that the patient is suffering from a progressive and incurable disease, when it is at least as probable that he is merely the subject of a malady which is eminently curable, is to adopt an attitude for which there is, and can be, no possible justification.

In May, 1905, a surgical colleague brought me a friend of his, who had been told by a very able physician that he was suffering from chronic Bright's disease. My friend, being a surgeon, was sceptical as to the diagnosis, and the patient, being a man of many responsibilities, was very impatient of the restrictions which the diagnosis entailed. He complained of occasional giddiness, breathlessness on slight exertion, and polyuria. There was a history of some "kidney trouble" twenty years before. He was now æt. 61 years. His urine was of sp. gr. 1012; it contained a definite amount of albumen. His blood-pressure was 200 mm. Hg., his apex beat was in the sixth space, half an inch outside the nipple line, and there was a loud systolic murmur in the mitral area. These findings seemed very strongly to confirm the diagnosis against which I was being appealed to, and I did not hesitate to say so, but I reserved my final opinion until a report could be obtained on the question of urinary casts. When this arrived I found to my surprise that, though a few hyaline casts were present, careful search had failed to reveal any of the more serious varieties. A second examination having confirmed this, I felt justified in venturing a more hopeful prognosis than that of the physician who had first seen the case. I did this, however, with considerable misgiving, for the albuminuria combined with the high blood-pressure and the cardiac enlargement seemed to afford evidence which no mere negative finding would be sufficient to controvert. The event, however, justified my faith in the negative finding. After three months' treatment, which included a course at a spa, the patient's heart had reduced itself to its normal dimensions, the albumen had completely disappeared from his urine, and his blood-pressure had fallen to 150 mm. Hg. He comes to report himself from time to time, but he has never had any further trouble and feels particularly well. His blood-pressure remains at 150 mm. Hg.—a figure which, at his age, can scarcely be regarded as excessive.

Let me now try and formulate the common denominator which I proposed at the outset. Albuminuria may be due to a great variety of causes, both physiological and pathological, of which structural disease in the kidneys forms but a small, if highly important, minority. The majority of the cases are due either to altered blood states or to failure in the normal vaso-motor mechanism. This failure may manifest itself in one of two directions. In the first, chiefly by some means so far undiscovered, the blood-pressure in the splanchnic area rises, and is maintained at a sufficiently high level to induce a renal plethora and consequent albuminuria. Of such are the cases of hyperpiesis of which I have just quoted an instance. In the second, owing to a local or general vaso-dilatation, the blood-pressure in the splanchnic area falls to the point at which a renal stasis is induced. Of such are the cases of cyclical, postural, and athletic albuminuria of which I have also cited instances, cases which for the most part occur in the young adults, in whom the vaso-motor response is either undeveloped, or, for some reason, inadequate.

Having regard to these facts, I venture once more to insist not only that, of itself, albuminuria affords no evidence of renal disease, but that, of itself, it does not present even a reasonable suspicion of the existence of such disease, any more than, of itself, dyspnœa presents a reasonable suspicion of cardiac disease.

*April 20th*, 1908.

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NICHOLAS DE PLANTIER has described the construction and use of the Bock-Samovar, an arrangement by which the hot water for a vaginal douche can be kept hot for an hour or more while it is slowly allowed to flow out. The heating is maintained by means of a charcoal block placed in a slide at the bottom of the douche-pan. This is lighted after the hot water has been introduced into the can and is kept alight during the douche.—*Med. Rec.*, vol. lxxiii, No. 13.

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# CONTUSIONS OF THE EYEBALL.*

By N. BISHOP HARMAN, M.A., M.B.Cantab., F.R.C.S.Eng.,
Ophthalmic Surgeon to the Belgrave Hospital for Children; Chief Clinical Assistant at the Middlesex and Moorfields Hospitals.

CONTUSIONS or bruises are of almost everyday occurrence; most of them are so trivial that we forget the bruise almost as soon as the initial pain of the blow that caused it has passed. Blows about the region of the eye are also common, particularly in certain quarters of the town on a Saturday night; happily most of these also do no more than cause the disfigurement of a black eye. But the eye is a very delicate and complicated organ, and blows that reach it are likely to result in some more or less serious disorganisation of its arrangements. It is of these that I wish to speak to you to-day.

Eyes differ greatly in their liability to injury by blows; a man with a good bony skull, with deep eye-sockets, and well developed brows and nose, has his eyes so well set in his head that they are protected from many an injury that might arise from blows of the grosser sort. On the other hand, eyes that are prominent, either by reason of the shallowness of their sockets, or else on account of a smooth brow or depressed nose, are particularly likely to receive the full brunt of any blow that may be aimed at them. The protection of the eye by the bony structures is, however, only efficient in warding off injury from large striking instruments; they serve but little as a protection against the injuries of smaller flying missiles. But yet in quite a large proportion of such accidents the eyelids, not the eyes, receive the direct force of the blow, and to some extent mitigate the shock to the globe.

Again, some eyes are healthy, others diseased, and as we should naturally expect, the effect of a blow upon a diseased eye is likely to be much more serious than it may be upon a healthy eye. By a good eye I mean primarily an eye that is well shaped, an emmetropic eye; its coats have not been thinned or put on the stretch by such a disease-process as myopia. Secondarily, I mean an eye that is young, an eye that is in all its parts healthily elastic, and without sclerosis of the vessels within or the fibrous coats without. A young, well-shaped, and elastic eye has all the chances of escape from injury in its favour; it can yield before the impact of a missile and regain its proper shape, whereas an older, harder eye would have been permanently altered by the bruise.

The variety of injuries resulting from bruises to the eye are almost limitless; the eye is so fine a piece of mechanism that a blow from quite a small missile, and one that from its nature can have but little momentum, is capable of doing irreparable damage. An attempt to give you anything like a complete description of all the possibilities of these injuries would be lengthy and wearisome; I will therefore hang my discourse on the events of nine recent cases, of which I shall show you drawings illustrating the conditions found. The injuries are those which are of most common occurrence, and, therefore, most to be kept in view.

The conditions following contusions of the eye may be grouped as they affect—(1) The anterior structures—iris, ciliary body, or lens; (2) the posterior structures—vitreous, retina, or choroid; (3) the particular liabilities of diseased eyes.

### *Blows affecting the Anterior Structures.*

CASE 1.—A boy, æt. 8 years, was shot in the eye by an arrow discharged by a toy spring pistol. The pistol is marked in large letters "Ideal," but it is by no means an ideal toy for babes. The projectile is a stick as large as a new lead pencil, the extremity is covered by an indiarubber pad. The spring is so powerful that it will project the stick sixty feet, and without the pad the stick pierced a dozen sheets of 'The Times' at a distance of a yard.

I saw the boy a couple of hours after the accident. From the state of the eyelids it was evident the padded end had struck the lower lid, so that the globe was bruised indirectly. The anterior chamber was full of blood—a condition known as hyphæma; the cornea was clear and bright, the conjunctiva chemosed, but the movements of the eye were good and the tension normal. It was clear the eye was bruised and not burst, but what internal damage the bruise had caused could not be determined; the blood obscured all view. After a rest in bed for a

* A lecture delivered at the Polyclinic.

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couple of hours the blood settled down to the lower part of the anterior chamber. The pupil could be seen to be oval in shape, with its long axis vertical; it did not react to light. Rest in bed, a smart purge, and atropine to the eye were followed by absorption of the blood in two days; the pupil was dilated and oval, now with its long axis horizontal, for the part least shaken by the blow had reacted best to the drug. There was no iritis, but a small fan-like opacity could be seen at the back of the lens, probably the effect of a jar to the fibres. The fundus was healthy, and with a lens correcting the hypermetropia $\frac{6}{9}$th vision was obtained. Recovery was speedy; the eye suffered no permanent damage.

The case illustrates the simplest effect of shock upon the vessels and muscles of the region. There is an instant paralysis of the local vaso-motor mechanism, and bleeding ensues; this is seen in the ecchymosis of the lids, the subconjunctival hæmorrhages, and the blood in the anterior chamber. The vessels of the iris are very numerous and very near to the surface, so they bleed easily. Next there is paralysis of the sphincter of the iris, or a paralytic mydriasis, resulting in an oval and immobile iris.

Case 2.—A barrister, æt. 35 years, was struck in the eye by a ball when playing squash racquets. The balls used are of pure rubber with a small central cavity; they are about the size of a golf ball. The ball had been served from behind him with great force; at the moment of delivery he looked round and received the ball on the lower lid. Happily the blow was not direct but glancing. I saw him an hour later. The lid was greatly swollen, the anterior chamber full of blood; there was bare perception of light when a powerful electric lamp was brought close to the eye. Examining the eye carefully with focal illumination a part of the iris could be made out, the pupil was dilated and fixed, and as I looked, on a sudden movement of the globe, the iris quivered; it was evident that the lens had been displaced, for the iris trembled from lack of support. Thereupon the blood covered the iris and no more could be seen. Three days later the blood was so far absorbed that the iris could be again seen; it was tremulous, and the margin of the displaced lens was visible; there was an opacity of the back of the lens similar to that noticed in the boy, but the posterior chamber was free from blood and the fundus was healthy. In a fortnight the only visible effects of the accident were an oval pupil, an iris slightly shaky in one part, and the mark in the back of the lens. The edge of the lens could no longer be seen. Distant vision with a cylinder was $\frac{6}{9}$, against $\frac{6}{5}$ with the good eye. After nine months these conditions remain.

The case shows that traumatic paralysis of the iris may persist; that the lens ligament may be torn, causing a slight subluxation of the lens, yet not so as to endanger its nutrition; that a small shaking of the fibres of the lens at the posterior pole may cause an opacity, either transient as in the boy, or persistent as in the man.

Case 3.—A parson was enjoying the physical recreation of wood-chopping. A splinter struck his left upper eyelid. The lid swelled and blackened, the eyeball was red and tender. Nine months later he came to me because he could not see well with the eye. The best vision was $\frac{6}{24}$ against $\frac{6}{6}$ in the other eye. On examining the media a number of radial opacities were found in the lens, and with the pupil dilated these marks could be seen to spring from one part of the edge of the lens, which was shrunken and cataractous. It was evident that a full half of the lens ligament had been ruptured. The rest of the eye was healthy.

Here was a more severe injury. The lens had been displaced to such an extent that its nutrition was affected, it was no longer clear, but shrunken and partly oqaque. Happily the lens remained sufficiently supported by the remaining ligament. Had the rupture been more extensive the lens would have sunk down on to the ciliary body, and constituted a foreign body in the eye, and in effect set up irritation, cyclitis, perhaps secondary glaucoma, or even induced that disastrous condition known as sympathetic inflammation. Such a sequence of events is the gravest risk involved in these injuries. However, it is safe to consider the liability is only present when the lens has been so far dislocated that it is in almost constant motion. Such a condition is inconsistent with useful vision of the eye, so that removal of the lens is called for, or failing this possibility, excision of the eye.

*Injuries of the Posterior Region of the Eye.*

Case 4.—A student was crossing the quadrangle of his college one snowy day when he

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received a snowball by way of salutation; unfortunately the aim was too true, he received the missile in the eye. He immediately found he could not see with the eye. There was the usual hyphæma; but when this cleared, vision was not recovered, for in the fundus over the macular region was a large subhyaloid hæmorrhage; this rendered direct vision impossible, but peripheral vision was obtained. With the slow absorption of the blood the central scotoma diminished, until it disappeared, and vision was restored.

Such an injury is usually set down to the effect of *contre-coup*. Personally I do not believe in such an effect in the eye. There is no analogy between eye and brain in this matter. A blow to the skull is not directly communicated to the brain within; the brain has its own inertia independently of the skull, and a blow to the skull sets up backward and forward oscillations between the skull and the brain mass, so that *contre-coup* is likely enough. The eye, on the other hand, is an elastic sphere completely packed by nearly homogeneous contents, and backed by a comfortable elastic bed. A blow causes no independent oscillations; if the front be driven in the sides and back bulge out to a degree proportional to the indentation. How, then, it will be asked, do these posterior hæmorrhages arise? Just as do the anterior hæmorrhages, by the sudden alteration of pressure on the vessels caused by the blow. Besides the retinal vessels at the macula are terminal vessels, and therefore, particularly liable to such injury, and in the region of the macula the hyaloid membrane is least firmly attached.

All hæmorrhages do not clear up so happily as did that in the student's eye; some produce irreparable damage to the retina or choroid, or bursting into the vitreous leave that medium permanently altered.

Case 5.—A woman, æt. 65 years, seen at hospital, was found to have excellent vision in one eye, but very poor vision in the other. At the macula of the defective eye there was found a small, oval, depressed area of one third the size of the disc, and with a well-defined edge. The condition is known as "hole in the macula"; it has been found to succeed upon blows to the eye, and it is a permanent damage. This woman had suffered a blow in that eye twenty years before, which may perhaps have been the cause of the defect.

I am inclined to think these holes are secondary to a hæmorrhage: the loss of proper blood-supply or the pressure of the effused blood may cause a necrosis of the thin tissue of the macula.

Case 6.—A boy was struck in the eye with a tip-cat, a toy that is useful enough to train hand and eye when used in a clear place, but an abomination in a street or crowded playground. When the boy was seen an abrasion of the cornea and all the signs of a severe bruise of the eye were apparent. The hyphæma cleared; and the iris was found partly adherent to a rather sunken and cataractous lens. Fingers could be counted, and tested by candle-light the field of vision was good. The opaque lens was removed by needling, and a clear pupillary opening produced. But when the vision was tested with an appropriate glass nothing better than $\frac{1}{60}$ could be obtained, for in the region of the macula was an irregular area of delicate superficial choroidal atrophy. It was doubtless one of the several effects of the bruise, and probably indicated atrophy succeeding to a hæmorrhage into the internal choroidal layers.

Case 7 illustrates a grosser form of injury to the choroid. A man was struck in the eye in a drunken tavern brawl. There was the usual black eye. Later he noticed that he could not see objects on the ground towards the left-hand side. Central vision was good. The field of the left eye presented a large blind sector down and out. In the upper and inner quadrant of the fundus was a long oval patch of chorio-retinal atrophy with crenated and deeply pigmented edges. The patch seemed to be the result of a large hæmorrhage into, and possibly rupture of, the internal choroidal layers.

### *Injuries to Diseased Eyes.*

There are two orders of disease which render the eye particularly liable to serious damage on very slight blows—angio-sclerosis and myopia. The damages likely to be sustained are just those that may occur spontaneously in these eyes; injury merely accentuates the liability, or provides the occasion for the occurrence. Sclerosed vessels are brittle, and liable to rupture; a bruise puts an extra strain on the weakened tissue, so hæmorrhages of a gross kind are found. In myopia, especially of high degree, there is a lack of agreement in the fitting of the coats of the eye; the progressive yielding of the sclera is not met by an equal

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stretching of the choroid and retina, so these more delicate coats are in an unstable state, and liable to be damaged, the choroid by rupture, and the retina by detachment.

Here are a couple of cases that illustrate these dangers well:

Case 8.—A young man with 6 dioptres of myopia knocked his left eye, and the sight was greatly reduced. I had seen him three years before, he then had $\frac{6}{6}$ vision in each eye. Now the vision of the injured eye was only $\frac{6}{60}$ with his glasses. In the fundus there was a horizontal split involving the macula, and stretching far to either side.

Case 9.—A young mother came to hospital with the story that a week ago her baby "was loving her," and put his tiny finger into her eye, and "the eye went dark." Examination showed a large detachment of the retina. She had 14 dioptres of myopia. Such cases are by no means rare.

### *Treatment.*

The one word *rest* sums up the main lines of treatment of these cases. Absolute rest, flat on the back in bed, is the prime essential.

Certain other points need care. Traumatic iritis is a likely complication. We may forestall this by the instillation of atropine to the eye, by the use of cold compresses to the eyelids during the first twelve hours (ice should not be used, the cold is too severe), and by the administration of a smart purge of calomel or even croton oil. If the inflammatory reaction be severe, local bleeding is of the greatest benefit, and this can best be secured by the application of a couple of leeches to the outer canthus, to be repeated as circumstances indicate. In subjects of a full habit a thoroughly old-fashioned phlebotomy is to be recommended.

To soothe a restless patient a draught of bromide of potassium and chloral may be relied upon. Slop diet is advisable for the first three days.

No rule can be given as to the duration of treatment; that must be decided by the circumstances of each case. But it is safer to err by tardiness in releasing the patient from confinement than to think too little of a serious injury.

### *Conclusion.*

Most of the cases I have brought to your notice were caused in play, a few in the course of work. Sport that does not involve some risk to life and limb in its execution would be poor sport, a mere pastime; most of us are content to take the risk. But we are bound to do what we can to enlighten the public on the folly of providing children with toys that are obviously dangerous to their possessors or to those near them.

At work there are other considerations. The employer is rightly held responsible for the injury to his man. It behoves him, therefore, to take every precaution to diminish the risks of labour, both for his own and for his man's sake. It is a small matter for a wood-chopper or a stone-breaker to wear goggles, but the protection may save heavy damages.

Lastly, part of our work lies in the exercise of the greatest care in noting the state of the eye when it is first seen and on every subsequent occasion. Only by such care can we be sure of our ground in diagnosis, in prognosis, in treatment, and in the not unlikely event of a cross-examination in the witness box during an action for damages.

*April 20th*, 1908.

**Posture to facilitate Delivery.**—Samuel states that a patient in labour with her first child was placed in a position so that forceps could be applied to aid the difficult delivery. She lay across the bed with her legs flexed on the abdomen while the nurse was bringing two chairs for her feet. At that moment a labour pain came on, and the woman seized her flexed-to-the-utmost legs just above the knees and pulled on them as she strained, the head at once making its appearance. This was the more remarkable, as the sagittal suture before had presented somewhat unfavourably and the labour pains had not had much effect. This posture evidently facilitates delivery, as it reinforces the expulsive action of the labour contractions, while at the same time the diameter of the pelvic outlet is enlarged. Since then Samuel has applied this posture in all his cases of difficult delivery, and has frequently thus been able to dispense with forceps. The woman pulls on her legs just above the knees, the thighs flexed to the utmost, and she raises her head a little at the same time. In frontal and transverse presentation space is gained for rotation of the head, and the posture is also unusually favourable for face presentation and with contracted pelvis.—*Journ. A. M. A.*, vol. l, No. 13.

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## A CLINICAL LECTURE
ON
## PNEUMONIA AND EMPHYSEMA.

Delivered at St. Bartholomew's Hospital.


By NORMAN MOORE, M.D., F.R.C.P.,
Physician to the Hospital.


GENTLEMEN,—There are three patients in Mark Ward on whose cases I propose to lecture to-day. Two of them you will see here, but the other is not quite well enough to leave the ward. They are examples of a very common disease, pneumonia. The first is a boy, aged 5 years, another is a boy, aged 14 years, and the third is a boy, aged 15 years. I want to mention one or two points in connection with all three, and then in particular to dwell upon a condition which is present in the third case, and which has a most important bearing on pneumonia, namely, emphysema of the lungs.

But first of all we will consider the three patients as cases of pneumonia. The boy, aged 5 years, is here, and he had pneumonia of the left lung, well marked. You may possibly be tempted to say to me, What do you mean by that? I am here using the word "pneumonia" not in any well-defined bacteriological sense, but as indicating a condition in which the lung or part of the lung becomes solid, with a very great rise of temperature and a continued condition of fever, which lasts about a week, and ends in a sudden fall of temperature. On January 30th this boy was at school and felt quite well. On that night, however, he became feverish and vomited. You know how frequent a symptom of rise of temperature in children vomiting is. The child was then brought to this hospital, and on January 31st, which was the first day of his illness, the temperature was 104° F. His temperature continued high till just before the commencement of the sixth day. And then, as you will observe from the chart, it fell suddenly, and it has continued normal since.

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The other boy who is here is aged 14 years. On February 1st he did his work until mid-day, but he felt ill on Sunday, February 2nd, and had rigors and shivering. He went out on Monday; but stayed in bed on Tuesday. He was admitted to the hospital on February 5th. He had therefore been ill three days before he came in, and on the sixth day of his illness his temperature underwent a sudden fall, and has continued to be nearly normal ever since.

The third boy, who is not well enough to be brought into the theatre to-day, is aged 15 years, and he was said to be quite well up to January 31st. At any rate January 31st was the first day on which he felt any illness. His temperature continued very high, and underwent a sudden fall, beginning at the end of the seventh day. It afterwards rose. I will ask you to consider for a moment his temperature chart. All the cases agreed in one particular, that they had a very high temperature for a number of days, and that then it underwent a sudden fall. That clinical history—a very high temperature beginning suddenly, with obvious pulmonary symptoms, and then a sudden fall—is characteristic of pneumonia, and it is very important to bear it in mind, because although when you begin to study the subject you think, "I can always tell whether a patient has pneumonia by listening to his chest," you will find as your experience enlarges that there are cases, particularly in children, in which you are unable to detect any physical signs which would justify you in saying that the case was one of commencing pneumonia. A sudden fall of temperature will be sufficient to make you certain that the patient has been suffering from pneumonia, and if on listening to the chest you have been unable, for other physical reasons connected with the conditions inside the chest, to hear the physical signs of pneumonia, if there is that sudden fall you can nevertheless be certain that it was present. So that sudden fall of temperature is a most important symptom to bear in mind You are sure when you are in practice to come across cases in which day after day for four or five days you remain uncertain whether the child—as this most often happens in children—has pneumonia, and then it has a sudden fall of temperature without a further rise. When that occurs, you know you have had to deal with a case of pneumonia. The case of each of the two patients here has a peculiarity of its own which is worth noting. This boy, aged 5 years, has a loud systolic murmur at the left base. This murmur is less loud at the apex, audible at the right base, audible in the left supraspinous fossa behind, and at the angle of the scapula, and the beats of his pulse are unequal. So one feels no doubt that he has disease of his mitral valve. He has had pneumonia, and I want to mention to you two or three points that ought to occur to you when you find that a patient with pneumonia has valvular disease. I will put the recollection of the least probable thing first. First of all you know that the pneumococcus of pneumonia has been discovered in the endocardium, and that there is no doubt that occasionally there may be an actual endocarditis due to its presence. Well, you say to yourself, is it probable that this is so? Of course, in the vast majority of cases you will be able at once to give a negative answer and say it is most unlikely. It is certain in this case, from the well-defined character of the murmur, which indicates some old change in the mitral valve, that it has not originated in that way. Secondly, you will ask what bearing has it on the chance of the patient's recovery? That is a most important point. I will take a case as far removed from this as possible in other circumstances, but in which valvular disease and pneumonia go together, and it is the kind of case of which everybody has seen a good many instances. I saw some time ago a medical practitioner, over 65 years of age, and he had pneumonia of his right lung, which was solid throughout. He had had for many years disease of his aortic valves. What was the effect? He died. Why? That was the combined result of the pneumonia and the valvular disease. Why did he die? Is a patient with pneumonia certain to die if he has pre-existing a valvular disease? The answer is "No": this boy is a well-marked example of that fact; he has recovered. In that practitioner there occurred a thing of which I have seen several examples when pneumonia in an old person was associated with aortic disease, and that is, the lung never cleared up. It never came out of the condition of solidification. The crisis in such cases arrives, the temperature falls but the lung remains solid; it does not clear up. The man, perhaps, coughs up something, but there remains absolute dulness, with bronchial breathing

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and bronchophony. And then one day, quite suddenly, the patient dies, as you constantly see reported in the newspaper, of "heart failure." What are we to conclude from that? We are to conclude that the most important point in the relation between valvular disease and pneumonia is, what is the state of the myocardium at the time of the attack? This old man whom I have mentioned had long had aortic disease. I have no doubt that he had extensive degeneration of the muscular tissue of his heart, his heart was therefore unable to do the work necessary to keep him alive, it was unable to keep up the pulmonary circulation in the way necessary for the clearing up of the lung, and ultimately it terminated his life by stopping. The important point when you are trying to estimate the prognosis in a case of pneumonia in which valvular disease was present at the time of the attack is, what is the condition of the muscular tissue of the heart? In other words, how much effect has the long-standing valvular disease had upon the heart? If the muscular tissue is sound, then the patient may recover, in spite of the valvular disease. If the muscular tissue is not sound, the patient is far more likely to die. There is another thing I want to say to you about valvular disease. What I have said applies to all forms, but if the patient has extreme constriction of his mitral valve, then that which is the most embarrassing form of valvular disease becomes still more embarrassing to the over-worked heart, and the patient may die, and frequently such patients do die, and not because the myocardium has undergone degeneration, but because the heart cannot stand any further strain upon it. If you look in old books you will find that they always recommend bleeding as the treatment for acute pneumonia. There is a famous text-book of medicine, Sir Thomas Watson's 'Lectures on the Principles and Practice of Physic,' a well-written book and which was for thirty years the chief text-book of medicine. In the first edition of that book, which came out in the year 1843, you will find that he recommended bleeding without the slightest hesitation for every case of pneumonia. And if you look at some of the old manuscript notes, some of which are in the library here, of Dr. Peter Mere Latham, of the twenty years before Watson, you will find that almost every patient with acute pneumonia was bled. Watson came, in later editions, to say that there were some cases in which it was perhaps better not to bleed. And that attitude of mind goes on until later still, for he cannot shake off his original belief: he says cases in which bleeding is useful are, perhaps, very few. And at last, in the fifth edition published in 1871, bleeding as a treatment for this condition is hardly mentioned at all. As far as we can guess from our present knowledge, we think it must have been hurtful in every case, that it really diminished the patient's chances of recovery in every case. Why did they continue to bleed patients with pneumonia? Of course we know that where an idea, believed to be a scientific idea, is accepted, it takes a very original man to see that it cannot be applied, and that therefore a rule based on it is wrong. But I have always thought that there must have been some cases in which apparent benefit occurred from the procedure. When you read Dr. Latham's notes—I have read a great many of them—you will see that, looked at from our point of view, the only effect of this bleeding was to make the patient's convalescence a great deal longer than it is at the present day. And if that was true, of course it means that his chance of recovery was less, and that he lost strength. It is possible that in cases where mitral stenosis was present—a condition they did not then generally recognise—that the bleeding did temporarily relieve the circulation, just as it would have done if the patient had not had pneumonia. A few cases of that sort may have kept up the clinical reputation of bleeding in the minds of very observant men. Of course, the majority of men accepted the general view that bleeding was the right thing to do, and there is no doubt that the enormous increase in the number of deaths was largely due to that procedure. I need not point out to you how important it is to keep your minds open as to whether what are supposed to be absolute truths in medicine are really absolute truths or not.

Now look at another point in the treatment of pneumonia. You will find that it was said by everybody for ages that patients with pneumonia must not be fed. Why? It was said that this was for the obvious reason that if you fed the patient you fed his disease and made the disease more powerful and the patient worse. That argument was accepted and handed down for perhaps two thousand years Nowadays we take

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a different view. We do not think that when we feed the patient we feed the pneumococcus which is in his body; we think we must feed the patient to enable him to maintain his strength while the pneumococcus is growing in his body. But you can see how assertions or dogma of that kind can affect treatment enormously. There seemed to be nothing improbable in the suggestion that by feeding the patient with the disease you were feeding his disease, especially if one did not know the nature of the disease. Our principle, of course, is to feed the patient throughout with the utmost care.

The other boy who is here illustrates another point with regard to pneumonia which deserves mention. Every pneumonia of the species I am describing, without exception, is a pleuro-pneumonia, that is to say, there is some exudation on the surface of the pleura; and if you look at the post-mortem examinations on all the cases of pneumonia which you see in six months here, you will see that there is not one in which the surface of the pleura is not more or less involved. And there are two points which occur to me about that in relation to this boy. First of all, remember that the degree of pleurisy, though it is often slight, may be very extensive; and it may be very extensive at the beginning. And it often happens that you may see a patient with raised temperature, flushed, with difficulty in breathing and a very intense pain in his side, worse on drawing a breath. You listen to his chest and you can hear friction sounds, and you say to yourself or to the patient or his friends, this is a case of pleurisy, and frame your prognosis accordingly. But two or three days later you find that the patient coughs up rusty sputum, and on listening to his chest you can no longer hear a friction sound, but you hear loud bronchial breathing and bronchophony, and you are convinced the case is one of pneumonia. It was really a case of infection of the lung as well as of the pleura from the beginning, but the colonies of micro-organisms in the pleura having developed and become powerful and numerous earlier than those in the lung, the physical signs of the pleurisy obscure those of the pneumonia, although the more important settlement after all was in the lung. This condition was illustrated in this boy. He had some signs of pleurisy, which were rather well marked: and a friction sound remained after his temperature had fallen. Of course, as there has been exudation on the surface of the lung, a friction sound may remain for a considerable time. It does not often do so, but it may. And the other thing which may occur, and which I have been on the watch for in him, though it has not occurred, is the formation of an empyema upon the side upon which the pneumonia was. That is worth remembering, because when the crisis has occurred and the temperature has fallen as you see in this chart, the medical attendant might not examine the patient's chest after that as minutely as before. You ought always to continue careful examination, as in youngish people empyema may succeed any extensive pneumonia and without very much notice. All that would happen to show its onset would be that the temperature would begin to go up, but there might be some collection of pus before there was any very marked change in the temperature, particularly if the temperature were not taken several times a day.

Those two cases are well worth considering in relation to the accompaniment of pneumonia by old valvular defects, and the relation of the disease to the pleurisy, which is always present in pneumonia at the beginning and at the end. At the beginning it may make the pneumonia obscure, and at the end it may lead on to empyema.

Of the third patient, who was not sufficiently well to leave the ward, I show you the temperature chart. You see from that that he has had a much more severe illness than have these two. The temperature fell on the eighth day, but rose again and went on. How is that to be explained? Of course if he had a large effusion with acute empyema that would explain it; but there are no physical signs in his chest justifying that view. Then what was the cause? I shall not dwell upon this, because I cannot prove it, but I think it is probable that the original part of his lung affected afterwards infected another part of the lung. That is a well-known phenomenon. I think there was a second development of the pneumococcus in another part of the same lung, or, more likely still, in the opposite lung. So the second attack is simply an infective pneumonia. Of course pneumonia may be conveyed from one patient to another, but that does not often occur, except where a husband has been nursed by a wife, or one sister has been nursing another, and there is

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very close attendance throughout the day. Instances of that everybody has met with, and in the Local Government Board reports there are instances of rather extensive epidemics of pneumonia in which there was a great deal of infection from one person to another. But the infection of one lung by the material from the other is not of very rare occurrence. That is what occurred here, and was the cause of this second rise of temperature. Whenever that occurs of course you feel anxious about the patient, because he has not cleared off the effects of one bacterial development before another is begun, and, therefore, he has two partly coincident developments of organisms going on in his system. But that was not the point I wished to dwell upon in this patient. When he came in his lips were purple, he looked dusky, he was breathing rapidly, and he was in a condition of urgent dyspnœa. On listening to his chest, though there was some impairment of resonance, there was not any absolute dulness on the surface, and there were abundant mixed râles to be heard, rather more on the right side, but certainly over both sides of the chest. In other words, the physical signs were those of acute and widespread bronchitis. On looking at his chest it was seen to be more or less rounded, less of an ellipse on section, more nearly a circle than it should be. There was no cardiac dulness. He had tympanitic hyper-resonance right down to the base of his lungs behind, so that he obviously had emphysema, though only fifteen years of age. Such cases will always cause you great anxiety, and it is in that relation that I wanted to say a little about emphysema. The etymology of a word is seldom of much use to us in medicine. We have inherited a great many words which were used when they were employed first in medicine in a totally different sense from that in which we use them, and, therefore, I only say anything about it because I want to impress upon you what the real meaning is that you are to attach to emphysema. In Greek, of course, the word means an inflation. It only occurs in a way capable of precise definition in one single passage in Hippocrates, where he speaks of dropsy. He says there are two kinds of dropsy; one is anasarca, the other is with emphysema (appendix to 'Regimen of Acute Diseases,' Littré ii, p. 496). He is obviously speaking there not of anything to do with air, but with dropsy, as you see it in acute nephritis, where, for instance, there is a rapid distension of the whole face of the patient. That was the first sense in which it was used, and the word merely means an inflation. Galen used it with regard to air under the tissues. He mentions emphysema as a collection of gaseous material under the skin or under the membranes covering the viscera. And as a result of that definition of his, in all books up to the most recent ones, a difference was drawn between "surgical emphysema," as they called it, and non-traumatic emphysema. Surgical emphysema was that in which there was air in the subcutaneous tissues following a stab, or the fracture of a rib, or other injury. It was, in fact, the emphysema described by Galen. Ordinary emphysema was a totally different thing. It was described in books as a dilatation of the air-vesicles. And the first thoroughly searching, though not very original, account of it was one written by the late Sir William Jenner in Reynold's 'System of Medicine' in 1871. He pointed out what he considered to be the different causes of emphysema. And in those you can still trace the influence of this old idea—that it was primarily air let loose under the tissues. For instance, they talked at that time—and it can still be found in books—of vicarious emphysema, that is to say, a condition existing where a bit of lung has been rendered solid or has been bound down by an adhesion in the neighbourhood, and is unable to do its work, and the vesicles have become enormously dilated. Again, if a man blew a trumpet or was a glass-blower, he had as a result dilatation of his air-vesicles, and so on. In all those views you can see the remains of this old Greek idea of emphysema. And I want, particularly in relation to this boy who is not here, to try to eradicate from your minds that view, and to lead you never to think of emphysema as anything but a degenerative condition. The man who blows a wind instrument or who is employed in glass-blowing, sooner or later, owing to the over work placed upon his lungs, and the fact that he ceases to have residual air, because he fills and empties his lungs each time, has lungs not equal to the strain and degeneration therefore takes place in the walls of the air-vesicles, so that they become bigger, and the lung is emphysematous. In people who have bronchitis and retain its products in the lung, that interferes with its inflation and nutrition, and emphysema results. I want you to bear in

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mind that it is a change due to degeneration, and having remembered that, that it is a most important degenerative change. I have not time to go into the pathological aspect of it, but I want to give you some suggestion as to the kind of way in which you can prove to yourselves that it is a degenerative change. Take an emphysematous lung in the post-mortem room. If emphysema exists in it the air-vesicles are dilated, and the lung will not weigh what it naturally should weigh—it weighs less. An emphysematous right lung may not weigh more than 16 oz., and an emphysematous left lung perhaps only 14 oz. The weight of the lung is always diminished in this condition, because something has gone. What has gone? The walls of the air-vesicles. The patient seems to have bigger air-vesicles, he has bigger cavities, but he also has fewer of them. In other words, he has less lung substance than is normal, and therefore less power of aërating his blood in the lungs. That is the invariable condition found in emphysema. You now see the importance of emphysema in relation to pneumonia. This boy, aged 15 years, had considerable solidification. What the precise nature of it was we could not tell, owing to its extensive bronchitic condition surrounding the solidified part; but a very large part of his lung was completely out of the circuit of the air, while his emphysema left him much less aërating surface than there would have been in an ordinary lung. That was the reason of the cyanosis which I mentioned, and that was the reason why during every day of his pneumonia we felt anxious, and thought it possible he might have such dyspnœa that he would die. He had this hyper-resonance, which I have spoken of, right down his back, and no cardiac dulness. Particularly always notice that in the young. Emphysematous people have lungs of less aërating surface, and far less elasticity. They breathe in dust, they cease to breathe it out again, and therefore the lungs become the place of settlement of organisms like the tubercle bacillus much more readily. A person under twenty who has well-marked emphysema is much more likely to be attacked by tubercle bacilli and die of phthisis than a person whose lungs are not emphysematous. Therefore always observe it carefully. And I will just mention to you one or two other instances in which it occurs. Often when you are called to see a patient with pneumonia you are told that he has often had attacks of asthma. Every patient I have seen who has had asthma has had emphysema; I have never seen a case of asthma in which emphysema was not present. Asthma, no doubt, is a spasmodic difficulty in breathing, but it is a difficulty which is not purely nervous; it is always associated with emphysema, and it may be associated with other changes too, but that is always present. Remember; therefore, that a patient who is asthmatical is in greater danger of dying of some lung disease than is a patient who has not asthma. I mention that, because many people have the idea that asthma, being a nervous condition, cannot affect the prognosis of lung disease in a patient. If you look at the lungs, in the post-mortem room, of the people in whose toe-joints urate of soda is found, in other words in cases of undoubted gout, in every case of that kind you will find that the lungs are emphysematous; the walls of the air-vesicles like the cartilages of the joints, have undergone degeneration. Then there are the vast number of cases which we see of chronic bronchitis—cases of old people who every year come into the hospital with lungs which are becoming more and more emphysematous.

What conclusion do I draw from this? I want to impress upon you the importance of thinking of emphysema as a condition profoundly affecting the prognosis of pneumonia. A patient between the ages of fifteen and thirty ought not to die of acute pneumonia unless he has some other lesion in his body. If he has renal disease the prognosis in his case is much worse. If he has valvular disease of the heart the prognosis is serious. If he has no other morbid condition he ought to get well. But in determining the prognosis people often overlook the emphysema, and yet that is the most important of all the anatomical conditions which make recovery uncertain in pneumonia. If the patient has extreme emphysema his living or not as the result of the pneumonia will depend on the amount of other lung which is not affected. He may not have enough respiratory surface to carry him through the week of pneumonia.

You should remember that emphysema is a degenerative condition, the progress of which you can do something to check. Every patient with gout has emphysema. You may absolutely satisfy yourself of that in the post-mortem room, and you should carry that information about with you, and, therefore, advise people who can afford it and who are gouty to take precautions against repeated attacks of the bronchitis, which will aggravate their emphysema. It is well worth while, long before those people notice any definite signs of lung disease, to advise them to take care as regards their chest in the winter. A great deal can be done to check emphysema by preventing the patient getting bronchitis. And the other point is, always to observe carefully in every case of pneumonia, whether the patient is emphysematous or not. If he is, you will be anxious throughout his attack, as we were with this boy, who, I am glad to say, is now recovering.

*April 27th*, 1908.

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# GONORRHŒA IN THE MALE, WITH SPECIAL REFERENCE TO TREATMENT.

A Clinical Debate held under the auspices of the Chelsea Clinical Society.

DR. J. H. DAUBER, President, in the Chair.

Mr. C. F. MARSHALL re-introduced the discussion, taking as his subject the treatment of chronic gonorrhœal urethritis. He said: I shall confine my remarks to the treatment of chronic gonorrhœal urethritis. It is, first of all, necessary to distinguish between chronic gonorrhœal urethritis and chronic urethral catarrh, but, as I shall endeavour to point out, the latter is often the effect of the treatment of the gonorrhœal urethritis. Let us take a case in point. A healthy young man contracted gonorrhœa six months ago, and was apparently cured in a few weeks. He was, however, advised to continue injections, and was still using them when he came to me six months later. Having made arrangements to get married the following week he was anxious to see whether he was cured of his former gonorrhœa. He first consulted an enterprising house-surgeon, who promptly passed a large sound. He afterwards had his urine examined by another doctor, who found "traces of gonorrhœa" and advised him to postpone the marriage ceremony indefinitely. This naturally upset him a good deal, and he came to me in a state of great trepidation. I may mention that this ill-advised patient had had no obvious signs or symptoms of urethritis for several months, not even the morning drop, in spite of continual injections and violent manipulations by the sound. The urine contained no gonococci or pus, and only a very few small epithelial filaments. It is hardly necessary to say that I advised him to proceed with the nuptial preparations. This was a case illustrating the evil, physical and moral, effects of too much treatment. It is necessary to bear in mind that the continuation of epithelial threads without pus or cocci is of no consequence, and is usually the effect of over-injection. It is common to see hospital out-patients continuing injections for months, or even years, simply on account of the persistence of a slight discharge. Of course as long as they continue the injections they are likely to have the discharge.

We have to consider the causes of chronic gonorrhœa. These are, first, neglect of, or imperfect treatment, in the acute stage. Secondly, too prolonged and too energetic local treatment. And thirdly, the presence of stricture. Fourthly, we have to consider differences in the seed and the soil—that is to say, the virulence of the cocci and the power of resistence of the patient.

First, then, the neglect of, or imperfect treatment in, the acute stage. There is no doubt that the prophylactic treatment of chronic gonorrhœa consists in thorough treatment of the early stages. Local treatment by injection or irrigation should be commenced as early as possible, as it is in this stage only that the gonococci can be successfully attacked. In a very short time the gonococci penetrate the epithelium and reach the sub-epithelial tissues, where they are not accessible to the action of injected fluids. Secondly, too prolonged or too energetic local treatment. When the cocci have reached the sub-epithelial tissues no amount of injection or irrigation will reach them. Moreover, the action of local remedies is still further impeded by the changes which take place in the epithelium itself. The cylindrical epithelium of the urethra becomes keratinised and transformed into stratified horny epithelium. These changes also affect the ducts of the glands of the urethra. How, then, can we expect the injected fluids to penetrate this hide-bound urethra and reach the gonococci which are effectually hidden beneath it? Motz has tried to get over this difficulty by using injections of protargol, hermophenol, glycerine, and cocaine, the injection being retained upwards of an hour by occlusion of the meatus. The glycerine is said to soften the keratinised epithelium and allow the penetration of the other drugs. The objection to this treatment is, that in chronic gonorrhœal urethritis the process, in the great majority of cases, is localised to certain portions of the urethra, usually the prostatic, and that this process of epithelial softening does not discriminate between the healthy and the diseased portions. The difficulty in the treatment of the urethra is that we are working in the dark; in most cases we cannot see what we are doing, not always even when using the urethroscope. Specialists in other departments often have the advantage over us in that

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they can sometimes see what they are doing. Let us review briefly the usual methods of treatment of chronic gonorrhœa: Irrigation by hydrostatic pressure; irrigation by syringe and soft catheter, deep injections with large syringe; instillation of nitrate of silver by Guyon's or Ultzmann's syringe; the passage of large sounds; massage of the prostate in cases of chronic prostatic gonorrhœa; urethroscopic treatment.

First as to *irrigations of the urethra and injections.* In nearly all cases of chronic gonorrhœa the process is localised in the urethral glands and follicles, and these are protected against the action of injected fluids by the hardened epithelium. All kinds of injections and irrigations appear, therefore, to be of little use. Not only are they almost useless, but they are actually harmful if prolonged for weeks or months, for they induce urethral catarrh, which itself renders the urethra more liable to fresh gonococcal infection, either from a fresh contamination or from auto-infection by rejuvenescence of the latent foci of gonococci. Professor Neisser, in his latest article, remarks that the continuance of irritating injections favours the growth of gonococci by maintaining serous infiltration of the urethral mucous membrane.

Next as to the *instillation of nitrate of silver.* The same objection applies to this, also the fact that we are working in the dark. How can a drop of nitrate of silver placed on the surface of mucous membrane affect a focus of cocci in the urethral glands whose ducts are plugged by keratinisation?

With regard to *dilatation by sounds*, this method is only applicable to chronic anterior urethritis, for the largest sound which will pass the penile urethra will not dilate the bulbous or prostatic. Some authors recommend gentle massage of the urethra over the sound, to express the contents of the follicles and glands. The objection to this is that gonococci may reinfect lacerations of the urethra caused by the passage of the sound.

*Massage of the prostate* is much advocated by American surgeons in cases of prostatic gonorrhœa, in which there are localised foci of cocci in the glands of the prostate. According to Jadassohn gonococci generally die soon when incarcerated, and everything should be avoided which disturbs them. In his opinion massage of the prostate often leads to epididymitis. Hence it may be wiser to let sleeping dogs lie. It may be objected that any focus of cocci may be the point of origin of general gonococcal infection. This is possible, but it is more likely that general infection will be caused by disturbing them than by leaving them alone.

With regard to the *urethroscope*, there are probably few instruments which have been more abused than the urethroscope. As the late R. W. Taylor remarked, it is to be regretted that it has been used very much as a toy, and has been to some simply a surgical hobby. On the other hand, it must be admitted that the urethroscope used in a rational way and in a conservative manner is useful in the diagnosis and treatment of some cases of chronic urethritis. It should only be used for simultaneous diagnosis and treatment, and should not be passed unnecessarily, and it should never be used in acute or recent cases. The urethroscope has advantages over other methods in that we can see the surface of the urethra. We, however, cannot see through it, nor yet see the gonococci which are lurking beneath it. And it is difficult to see with it the posterior urethra. The urethroscope is useful in the treatment of chronic inflamed glands of Littré and the lacunæ of Morgagni in the anterior urethra by means of nitrate of silver. In all instrumental treatment of the urethra we have to remember that we are dealing with a sensitive and damaged mucous membrane, and that we can only imperfectly observe the extent of that damage. As the late Sir Henry Thompson said, "The introduction of an instrument is, more or less, an evil never to be resorted to unless a greater evil be present which its employment might possibly remedy."

In cases of gonorrhœa combined with stricture it may be truly said that in the treatment we are between the devil and the deep sea. We must dilate the stricture, but in doing so we are liable to open up channels for deeper gonorrhœal infection. In such cases dilatation should be combined with irrigation.

Lastly, I will say a word about the soil and the seed, about differences in the virulence of the cocci in various cases, and the resistance of the patient. The resistance of the patient probably varies much more than the virulence of the cocci, and this leads us to the question of acquired immunity and the question of inoculation. It is often stated that an attack of gonorrhœa confers

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no immunity against another attack. But this is not in accordance with the results of clinical experience, which show that the first attack of gonorrhœa is nearly always the most severe, and that if the person has subsequent attacks they are of diminishing severity and frequency. It is common knowledge that "hardened Don Juans and weather-beaten prostitutes" suffer less often and less severely from gonorrhœa than do the neophytes of both sexes. Hence there is an acquired immunity or natural vaccination. A French author has recently gone so far as to suggest that the best treatment for gonorrhœa, after having once contracted it, is to contract it again as often as possible. This brings us to the question of inoculation or vaccination against gonorrhœa. Cases have been recently reported in America of gonorrhœal urethritis treated by inoculation with cultures of gonococci. Sir Almroth Wright has gone still further and practised inoculations in cases of gleet under the control of the opsonic index, and his main point is that gleet is due to other organisms than gonococci, and that it is necessary to discover the organism in the discharge and inoculate against it. It is too early yet to express an opinion as to what is the value of this treatment. The conclusions I have come to are these: That the best preventive of chronic gonorrhœa is early and thorough treatment of the acute stage. That all forms of local treatment in chronic gonorrhœa are more or less unsatisfactory. That many of the complications of chronic gonorrhœa and many cases of chronic urethritis are caused as much by excessive local interference as by the gonococcus itself. That in all cases of chronic gonorrhœa the power of resistance of the patient should be increased by general treatment, especially when he is suffering at the same time from other diseases, such as syphilis or tubercle.

Mr. CHARLES RYALL: First of all, Mr. President and gentlemen, allow me to thank you most heartily for the honour you have paid me in inviting me to take part in one of your well-known clinical debates. I assure you it is an honour which I very much appreciate.

When I was asked to participate in this debate your secretary sent me a list of those gentlemen who were also taking part, and the subjects which they proposed to discuss. It apparently left little for me, as I found one of them had to discuss the treatment of gonorrhœa in its acute stage; another the treatment in its chronic stage, and a third was to speak concerning its complications. It required some consideration to decide what I was to do. Fortunately I was the last speaker taking a leading part, so I thought I would restrict myself to some debatable points in the treatment.

Well, sir, there are numerous opinions as to the correct treatment of gonorrhœa, but no man has a monopoly of intelligence, and I certainly do not claim to have such. Therefore, I hope that in approaching this subject to night, you will not think that I am doing so in any narrow-minded way, for, after all, conviction as to the proper line of treatment is mainly the result of one's experience, and I can only give you the conviction which is the outcome of my own experience in regard to gonorrhœa.

There are two schools: those who advocate the expectant, and those who advocate the active treatment. Active treatment is divided into various kinds: the abortive, semi-abortive, etc., but we will class them together for the occasion.

In the expectant treatment the patient is put to bed and general medical measures are adopted. No local remedies are either recommended or resorted to, because local applications are considered mischievous, and, to the advocates of the expectant treatment, they are regarded as mischievous in the extreme. The expectant treatment, therefore, more or less, hopes for the disease to undergo natural cure. On the other hand, the advocates of the active treatment believe that the proper way to treat gonorrhœa is by the application of remedies to the affected part, the disease being attacked in a more or less energetic way with a view of curing the infection.

Personally, I am an advocate of the active treatment, and from my experience of the disease, this is the form of treatment which I unhesitatingly recommend.

First of all, let us consider what is gonorrhœa? It is an infective inflammation of the urethra. It begins in the urethral mucosa, immediately behind the fossa navicularis, and tends to spread backwards as well as into the sub-mucosa. It does not remain localised, but spreads backwards along the urethra, and this backward spread is its natural course, and is not due to local applications, as the

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advocates of expectant treatment would lead us to believe.

In its backward spread it comes to the compressor urethræ, and it is the belief of many that this muscle offers a natural barrier to further spread of the disease into the posterior urethra. But how can the compressor offer any obstacle to the march of the gonococci? The mucous membrane of both the posterior and anterior urethra are continuous, and, as gonorrhœa is an infective inflammation of the mucous membrane, the compressor urethra can offer no more resistance to the spread of infection than can an elastic band, as is sometimes recommended as a measure in preventing backward spread.

Gonorrhœal infection, in the natural course of events, tends to spread backwards, and in consequence of this the whole urethral canal, and not one part alone, must, for all practical purposes of treatment, be considered as the possible site of disease. Clinically, the backward spread is not generally recognised, because by the time the gonococci reach and infect the posterior urethra their virulence in many cases has, to a large extent, abated. Infection of the posterior urethra may therefore take place without giving rise to any very acute symptoms or marked evidence of its presence.

It is this liability of such common infection of the posterior urethra which necessitates the whole urethral canal being considered the site of infection, as far as the treatment is concerned. Finger estimated that in 80 per cent. of his cases the posterior as well as the anterior urethra becomes infected, and from my experience I have no reason to believe that he has in any way over-estimated what takes place.

We have therefore, infection which tends to spread back to the posterior urethra, and it is my experience that passive treatment will not prevent this extension of the disease. The fact that the patient is taking active exercise may hasten and lead to early infection of the posterior urethra, but lying in bed will not prevent it. Therefore, what treatment are you going to adopt? What treatment would you adopt for a similar infection in any other part of the body? Will you simply recommend rest, and trust to the infection dying out?

It is a spreading infection, and therein lies its gravity. It is for this reason mainly, that I am a strong advocate of active measures, and I am convinced that it is the proper treatment to adopt, from the commencement of the disease.

One of the questions I would like to discuss to-night is the value of local remedies.

In the treatment of gonorrhœa, how is it that the expectant treatment has become, to a large extent, popular? And why is it that active treatment has fallen into disgrace with a certain proportion of the members of our profession? I think that, to a great extent, this is due to measures having been adopted which were improper, many kinds of injections having been used in the past which were harmful in the extreme.

It is essential that you select a proper local remedy, and it is worse than useless to apply anything that is likely to so affect the uninfected mucous membrane as to lower its vitality, and thereby hasten or encourage the spread of infection.

In the past strong remedies have been used—remedies which, owing to their astringency, have checked the discharge to a certain extent, and, therefore, like the ostrich, which buries its head in the sand, the men who used them were led to believe that everything was going on well. But remedies of this nature, which apparently give temporary relief, frequently hasten the spread of the disease by damaging the healthy mucous membrane, and they are, in a large measure, accountable for the present-day prejudices against active treatment.

Therefore, one has to think of what are the remedies to use. It is necessary to put them into two classes: those to be used by the patient, and those to be used by the medical man. I will say little about the former, except that, as far as the therapeutic value is concerned, you cannot possibly compare that of self-applied remedies with those applied by the medical practitioner; in fact, it is only in the hands of the practitioner that local remedies can be satisfactorily used.

As a local method of treatment, I am a strong advocate of irrigation, and I am of the opinion that it should be applied as early as possible in the course of the disease.

Having listened to what was said at our last meeting about irrigation, I had the impression that there seemed to be some misconception as to what was meant by this method of treatment. When I refer to irrigation I mean the washing out of the lower

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urinary tract, without the use of any instrument passed into the urethra, without a catheter, and with only the aid of a nozzle fitting the meatus.

Irrigation is accomplished by knowing how to make the patient relax the compressor urethra. It does not require any great pressure of fluid, and, in fact, as long as the irrigator is at least one foot above the patient's hips, irrigation may be carried out. Placing it higher than this is not conducive to the patient's comfort or welfare.

Irrigation can be very easily accomplished. It requires no great skill, but it requires a little practice. By this method the whole lower urinary tract, including the posterior and anterior urethra, and even the bladder, can be satisfactorily flushed. In difficult cases, when the compressor cannot be readily relaxed, this difficulty can be overcome by the application of cocaine.

I do not advise irrigation by means of the passage of instruments. Some advocate carrying it out by means of a catheter passed into the anterior urethra, others into the posterior urethra, and some even into the bladder. But it can be easily accomplished by the simple method to which I have referred. Moreover, the passage of instruments is always fraught with a certain amount of danger.

Now, as to what irrigation does. Take an ordinary patient, with a thick purulent discharge, much pain and scalding, and even with acute infection of the posterior urethra, and, perhaps, chordee. One irrigation itself, with a proper solution, will reduce the thick purulent discharge to a thin watery one. It will relieve his pain and chordee, and also the strangury arising from the posterior urethritis. No other treatment can do so much in such a short time. I think that this amount of relief also justifies one in carrying out this treatment. And not only does it relieve these urgent symptoms, but, where posterior urethritis is not yet present, it is my belief that it prevents infection of this part of the canal, and the consequent liability to epididymitis, and, moreover, it tends to an early cure of the disease.

What solution should be used for these local applications, especially by means of the irrigator? Personally, I think there is nothing better, in the acute stage, than dilute solutions of permanganate of potash. And the next best remedy is either hot boracic solution or hot water.

The irrigation should be carried out frequently—at least two or three times a day—if one expects to have early recovery or any rapid result from the treatment. While this is being done rest is essential, as exercise is exceedingly harmful for this condition, and will not only retard recovery, but will conduce to the developement of complications, arising from the spread of infection.

As to the evil consequences of irrigation, of which one has heard a good deal, you need not consider them seriously. We are told that it causes backward spread of the disease, but, as I have explained, some authorities almost lead one to believe that the gonococci make an onward march as far as the compressor, where their further progress is barred by this natural barrier. They are, metaphorically speaking, knocking at the door, but are unable to gain an entry into the posterior urethra. At last their opportunity comes, when some advocate of the active treatment pushes them through by means of irrigation, and thus infects the posterior urethra. I maintain that infection arises without this, and that irrigation prevents it.

I have never, myself, seen any ill effects following this method of treatment when it is properly carried out, and yet, personally, I have used it some thousands of times. And at the Lock Hospital it has been used many thousands of times more.

Apart from irrigation causing infection of the posterior urethra, it is also supposed to cause infection of the bladder. But infection of the bladder in gonorrhœa is exceedingly rare. Much confusion seems to have arisen between the diagnosis of posterior urethritis and inflammation of the bladder. The symptoms are so very similar that where a violent attack of posterior urethritis comes on, with the usual frequency of micturition, strangury, and pain, it is frequently mistaken for cystitis. As a matter of fact, it is exceedingly rare for gonorrhœa to spread to the bladder and cause cystitis, although I admit that there are occasions when it does occur. The majority of these are when, owing to instruments being passed, infection has been carried into the bladder, and the bladder, being injured by these instruments, the gonococci have thus been enabled to grow and cause cystitis.

Another debatable point in the treatment of

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gonorrhœa is as to what should be done when complications arise. The commonest complication is posterior urethritis and its onward spread to the epididymis.

I was taught in my early days that when the infection spread so far one was to stand off, and that active treatment was to be discontinued.

Well, sir, I must say that I advocate active treatment when such a complication as posterior urethritis arises, and I also advocate the same treatment when epididymitis is present. You have only to think what are the possibilities of infection of the posterior urethra. The disease so readily spreads to the adnexa, where it may become difficult to cure, and where infection may lurk for many years and be a source of danger, not only to the individual, but also to others.

If there is any occasion during gonorrhœa when active measures must be adopted to cure the disease, it certainly is when the posterior urethra is infected. And if it refuses to respond to irrigations with permanganate of potash I always advocate applications of nitrate of silver solution, gr. 5 or gr. 10 to the ounce, to the posterior urethra.

We are told that it is absolutely wrong to interfere on these occasions, but this is the reverse of my experience.

Also, in epididymitis active measures are again indicated, and again, irrigation gives most satisfactory results, and is generally followed by a relief of pain, tenderness, and swelling. This can only be accounted for by the curative action of the irrigation of the posterior urethra, the *fons et origo* of the infected epididymis.

In very intractable cases severer measures have to be adopted, and here, again, I believe in the application of nitrate of silver solution to the posterior urethra. While much infection persists in the posterior urethra, epididymitis is always slow in clearing up, and is also liable to relapse.

As to applications to the inflamed epididymis, I think that they are of little use, excepting applications of heat and cold. The same may be said for treatment by medicated suppositories, not only in epididymitis, but also in infection of the posterior urethra and its adnexa. How they are to act it is difficult to know, and I am certainly at a loss to explain. In fact, I think you may just as well put a bismuth plaster on the epigastrium in a case of gastric ulcer, with the hope of relief, as to expect a medicated suppository in the rectum to have any effect on the posterior urethra.

For the inflamed testicle, rest, application of heat, and elevation of the inflamed part, combined with local applications to the posterior urethra, give most satisfactory results. And, if anything further than that is required, I must say that I have, on many occasions, found a modified Bier's treatment, as recommended years ago by Valentine, of New York, as being most efficacious. Bier's treatment is now used in some acute infective troubles, but years ago Valentine found that a constricting band of adhesive plaster placed above the inflamed testicle had most beneficial results.

Now, as to the persistence of gonorrhœa. It tends to cling more tenaciously to the posterior urethra and its adnexa than to the anterior urethra, and, in my experience, the commonest cause of gleet, or prolonged discharge, is undoubtedly from the posterior urethra.

The treatment here covers a tremendous amount of ground. We have listened to a very thorough and learned discourse from Mr. Marshall to-night, who went over this part of the subject in a very complete manner, reviewing modern thought and modern research, so I do not propose to go extensively into this part of the subject. But, as far as treatment goes, I again say that it is my opinion that energetic measures should be adopted here. Irrigation is most valuable, but somthing more than irrigation is indicated in many of these chronic cases. Where irrigation fails, or in cases of long standing, one must resort to something stronger, such as local application of the silver salts, or even sulphate of copper, a remedy which is much used in the clinics in Vienna.

I cannot say that I know much about the serum treatment of the disease, but I agree with Mr. Marshall that in some cases you find that where there are successive attacks—and some people have many attacks—the later ones are much milder than the earlier. It is astonishing how some people seem to contract repeated fresh mild attacks of the disease.

As to the value of the endoscope, I think that in some conditions it is of great value, but its value is very much over-estimated. I look back to my early days of endoscopic work and I wonder now at the many things which I imagined that I saw; but time and experience have altered my opinion. In some

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hands the endoscope is but a plaything. At the same time, I can only say that in questions of diagnosis, and especially where there are complicated strictures, the endoscope is of great value.

I much deplore the fact that the public have been taught to look upon an attack of gonorrhœa as a very mild, harmless, and innocent sort of thing, to be treated with levity, but, as a matter of fact, it is an exceedingly serious disease, and its gravity should always be explained to the patient. Once it is present every measure should be adopted to eradicate it, and also to see that it is really eradicated. It is the cause of great suffering, not only to the male, but, as you know, sir, it is the cause of terrible suffering in the female; and not only you, with your extensive experience, but the merest tyro in gynæcological and abdominal work knows how serious are the results which follow gonorrhœal infection of the uterus and adnexa.

In the female it is often difficult to detect, difficult to treat, and more difficult to cure. I think we ought always to impress on our patients what a serious disease it is, and the dangers that follow an attack, especially in the female. We should impress upon these people that unless we are absolutely certain that there is no evidence of the disease left, they should on no account enter into the bonds of matrimony.

The CHAIRMAN expressed the hope that the points which had been so ably brought out by the speakers would be fully discussed by the meeting in the time available. A good deal of difference of opinion had been expressed by the four gentlemen who opened the discussions on the two nights, and it was very difficult to those who had listened to the views to make up their minds as to what was the best line of treatment to adopt. As he listened to Mr. Marshall, he felt convinced that too much activity was detrimental to the patient. When, on the other hand, he heard Mr. Ryall, he felt convinced that the expectant treatment must be entirely wrong, and that the disease could not be treated too energetically or vigorously. He thought the solution was that each case must be treated on its merits. But all the speakers seemed to have insisted on the importance of early treatment in the disease. Cases of gonorrhœa must be tackled early if there was to be hope of speedy cure. They could not be dealt with too soon. He believed Janet's method of treatment quite superseded the old method by the syringe and strong injections. He once had an American patient who came and asked him to carry out that treatment for him. He was almost a "martyr to gonorrhœa." He was a traveller, and said he never came to London without getting gonorrhœa. His attack was severe, and after each irrigation he gave a sigh of relief and said he felt very much better. The mere fact of washing out the urethra with hot permanganate solution gave him infinite relief, and he got well in an extraordinarily short space of time. He (Dr. Dauber) had no idea it was so effective, and he could confidently recommend it to those who had never tried it. He would have liked to have heard the speakers dwell at greater length upon the question of marriage in association with gonorrhœa. As a gynæcologist it appealed to him as a most important question. The doctor was often called upon to tell a young man who had had gonorrhœa when he might marry. There could be no doubt that many young men married who ought not to do so. They believed they were cured, and went to their doctor, who also believed so, but in a year or two after marriage their wives were found to be infected with gonorrhœa, and suffered from salpingitis, endometritis, and pyosalpinx, with the result that they might be quite crippled and might be rendered sterile by the pathological process, or they might have to undergo a serious operation. Within the last fortnight, at the Hospital for Women, Soho Square, he had had to operate on a married woman who was perfectly innocent and faithful to her husband, but who had been infected by her husband. And one said what a brute such a husband must be, and what hard lines upon a woman who married such a man, believing him to be healthy. But often the husband had put off marrying until he believed himself cured of his gonorrhœa, and perhaps had taken advice from his medical man, who also believed him to be cured. He thought it would have been a good thing if some of the speakers could have laid down some directions for the guidance of general practitioners so that they might be able safely to say to a man who had had gonorrhœa: "You may safely marry without any fear of infecting your wife." Because the wife in her turn might infect the child, which might suffer from ophthalmia neonatorum. It was

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very sad indeed to see women with their lives and their health ruined by an attack of gonorrhœa acquired, one might say, in the discharge of their duties. He would also have liked to have heard a ruling on the question of taking alcohol in the course of an attack of gonorrhœal urethritis. Men in this country were so fond of their drinks that often one of the first things said to the doctor was: "Cannot I drink something? Not even one whisky and soda?" Was alcohol banned, or should they be allowed some alcohol? Sometimes a man thought himself cured of his gonorrhœa, and then had a drinking bout, or went to some dinner and had a few glasses of champagne, and that seemed to bring back the symptoms again. With regard to the relative value of the active and the passive treatments of gonorrhœa, he might say that when he was abroad, in his youth, he saw worse cases of gonorrhœa and syphilis than he had ever seen since; the cases were in men who lived three and four hundred miles away from the centres of civilisation, and a very long way from any doctor, such men as those who were up river, on coasting ships, etc. In some cases they were eaten up with syphilis and gonorrhœa, with bad strictures and various other complications. One of the men of his acquaintance lost his life in consequence of a bad stricture, because he had no catheter and no one to relieve him. Therefore he thought the expectant treatment, if carried very far, was not advisable.

Dr. Foster Palmer said he could not throw much light on the subject, because in the present day he rarely saw any gonorrhœa at all. Twenty years ago he used to see cases, and with treatment they generally got well. But in those days but little was heard about irrigation. It was a question then of either what was now called expectant treatment—*i. e.* doing nothing—or using mild injections at an early stage, or strong injections at a later stage, with the view to cutting the course of the disease short. Their early use was supposed to set up violent inflammation. Irrigation came on at a later date. He had come across many cases in which the man had had gonorrhœa, appeared to be quite cured, had married, and his wife apparently had been free from everything up to that time, but all the rest of her life she suffered from what was supposed to be leucorrhœa. So often had that been the case under his own observation, and so often had it occurred in the practice of others, that he had sometimes doubted whether gonorrhœa was ever really cured, whether a man who had had the disease once or twice was ever really so free from it that it was impossible for him to infect his wife. He supposed that with the present bacteriological knowledge and research that question would now be set at rest definitely. He would be glad if the openers in their reply would state their views on that matter. Mr. Ryall's statement that in nine cases out of ten the gonorrhœal inflammation spread to the back of the urethra was new to him, because most of them seemed to have the impression that in an attack of gonorrhœa it was only the anterior portion of the urethra which was attacked, and that it was only by some accident or bad treatment that it extended to the posterior urethra. But, as Mr. Ryall said, the gonococcus did not crawl along the urethra, but spread in the mucous membrane itself, the various parts being infected by contagion. But if that were so, why was it that in nine out of ten cases the gonococcus did not infect still further and cause cystitis? And in view of the numerous remedies which had been suggested, he would ask about one which he had not heard mentioned, but which had produced magical effects in cystitis, and would probably have a good effect in gonorrhœa, although he had not had cases to try it on. He referred to methylene blue. He had seen cystitis, which was intractable to everything else, cured by its means.

Mr. Wyndham Powell said that with regard to the abortive treatment in the acute stage, if the man came on the first day within a few hours of the initial symptoms, nitrate of silver should be used for the anterior one and a half or two inches of the urethra of a strength of 5 gr. to the ounce, applied thoroughly with a small syringe six or eight times, taking one or two minutes over the proceeding. The patient grips the penis with the finger and thumb at the proper spot. In a number of cases the disease would be aborted by that method. If the disease were between one and two days old when seen, two inches or two and a half inches of the urethra should be treated with a stronger solution, 7 or rarely 10 gr. to the ounce, and the whole anterior urethra irrigated with 1 in 2000 permanganate as well. He does not usually

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repeat the silver, but begins the irrigations, if necessary, on the following day. In the case of suspicious intercourse, before the appearance of any discharge, the 5 gr. to the ounce treatment should be applied for one and a half inches, and then there would certainly be no attack. If the discharge had been in existence more than a couple of days it was better to commence with the permanganate at once. It seemed almost unnecessary to say now that the irrigation method of treatment was the only one for acute gonorrhœa, and the sooner it was commenced the better. If the patient came with a discharge, and there was uncertainty as to whether it had existed three, four, or more days, the anterior part was washed out thoroughly with hot water, using a couple of pints with a good pressure and then let him pass water, and see if there was anything in the posterior urethra. If not, the disease would be kept in the anterior part if the irrigations were used properly. He was sure the height of a couple of feet for the irrigator can was not enough. He was in the habit of using as much pressure as he thought the compressor muscle would be able to resist. It was possible to balloon the urethra fairly rapidly and in that way to put the muscle on the alert, having the patient standing up. Then five or six feet pressure could be used. The nozzle is put into the meatus, and with the finger and thumb of the left hand closing the meatus the pressure could be estimated better than in any other way. It was a sort of safety valve, and there was no need to give full pressure by closing the meatus absolutely unless it was so desired. The ballooning of the urethra was obtained by closing the meatus rhythmically about twice a second. In that way the infection could almost certainly be prevented from spreading to the posterior urethra. Posterior urethritis was not a complication; the disease spread there in the usual way from 70 to 90 per cent. of the cases. Before he did so much irrigation, and since, he had never neglected to wash out the anterior urethra with a pint or two of hot water to see if the posterior urethra was affected; it could be determined by the slight mucus or shred in the urine which was passed. In this way it could be diagnosed at least twenty-four hours earlier than by the two glass test. If the disease was commenced to be treated before it had passed the muscle it could be prevented from getting beyond it. No instrument should be used. He had had two cases lately in which the catheter had been passed to irrigate the posterior urethra in gonorrhœa, and in both of them acute prostatitis had been set up. If the disease had spread to the posterior urethra, he did not forget that unless he could have the man at rest he would not irrigate the posterior urethra in an acute case and let the man walk about, because of the liability to prostatitis. It is of the utmost importance to recognise the posterior invasion early, for, like the anterior, that is the time to abort it. If the patient was at rest he would use a little 2 per cent. solution of cocaine, putting 20 to 30 minims in with a small syringe, or eye dropper, rubbing the bulb in a backward direction (to force it into the posterior part) underneath, and allowing it to remain a couple of minutes, and then irrigate from a height of four or five feet with the patient on a couch. In that way no irritation was caused in any part from excessive force of the irrigation. The strength of permanganate he generally used for anterior irrigations was 1 in 1500 once daily. He did not use it weaker unless there was some special cause for doing so. Simpler solutions were boracic acid and mercury oxycyanide, 1 in 5000. In irrigating the posterior urethra, if the prostate was involved, he would not do it at all until the prostatic inflammation had practically subsided, and then he would employ 2 per cent. solution of cocaine, and irrigate with weak permanganate from a height of four feet. In hyper-acute cases of anterior urethritis weak solutions should be used twice daily. The vast majority of patients with gonorrhœa recovered perfectly nowadays. A certain number had residual gonorrhœa, lasting for many years. He had recovered gonococci from a patient six years after the acute attack, the man having had no questionable connection at all in the meantime. He had one some time ago in which the man went through the South African war, and had not any connection in the meantime, and there were many gonococci from the prostate four years after contracting the disease. If there were no shreds from the anterior urethra and massaging the prostate very thoroughly and collecting the secretion and microscopically examining it, and that were repeated seven or eight times with a negative result, one could say that there was no danger of infection should the patient marry. A patient came to him

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a short time ago from the co intry. He had recently been married, and had given his wife the disease. But he had consulted six men in and about his town to make sure, and they all pronounced him sound. The cause of the infection was that he had a minute sinus by his frænum leading to a residual gonococcal abscess. With regard to the value of the urethroscope; in cases which were getting somewhat chronic one very great advantage was to be able to see that there was no stricture or disease in the bulbous part. If the anterior three or four inches only was involved in the chronic process it was absurd to pass instruments into the bladder as is commonly done. The necessary dilatation could be done with a short, straight sound, or by means of a Kollmann's anterior dilator.

Dr. SEYMOUR MAYNARD said he had a case of gonorrhœa, and after massage of the prostate and cultivation of the slides he could not discover the gonococcus, but only the xerosis bacillus. He would like to hear what connection there was between the two, if the discharge kept on. The specimens had been sent to various laboratories, but the gonococcus had never been discovered, although the massage had been very complete by specialists.

Mr. MARSHALL, in reply, said it was difficult to define certain tests for marriage, but so long as the threads obtained were simply epithelial, and there was no pus and no gonococci after several examinations, the patient could safely marry. Alcohol, he thought, should be avoided in the acute stages of the disease: but in chronic cases he thought a little was admissible. With regard to the question of the comparative infrequency with which the bladder was affected, he supposed that one reason was the acidity of the urine, which inhibited the growth of the gonococci. The President had remarked on the apparent discrepancy between some of the statements he (Mr. Marshall) had made, and those of Mr. Ryall. But Mr. Ryall was speaking of treatment of the acute cases, and in that respect he was in agreement with him. He (Mr. Marshall) was referring to the inadequacy of most methods for treating chronic cases, and the disappointing results which were obtained in them.

Mr. RYALL, in reply, said the cure of the disease in the male was comparatively easy compared with the cure of it in the female, and therefore it was imperative for one to see that all cases of infection in the male were cured before marriage took place.

In the female gonorrhœa was difficult to both detect and to treat. The cases in which it was difficult to know whether the patient was free from infection were not those in which the urethra epididymis and prostate became affected, so much as those where the vesiculæ were the seat of infection. It was in cases of gonorrhœl vesiculitis, where infection often remained hidden, and where gonococci could be found only after repeated massage. Neisser insisted upon examining the contents of these organs before he was satisfied that the patient was free from infection. With regard to gonorrhœa, he was a temperance advocate. The doctor should strongly urge the patient with gonorrhœa not to take alcohol. He gave that advice, probably, because of the irritation produced, which encouraged the growth of gonococci. In fact, alcohol was a well-known test as to whether a particular individual was cured. If he were able to take alcohol, especially beer and champagne, he was supposed to be cured.

Answering Dr. Palmer, he said most practitioners had come across cases in which the gonorrhœa was supposed to be cured, but in which, some time afterwards, owing to gonococci lurking in some back-water, in the prostatic follicles, or vesiculæ seminales, and for some reason again becoming active, the disease breaks out afresh.

The question why the bladder was not more often affected had been answered by Mr. Marshall. It was extraordinary to find how immune the bladder was.

Methylene blue had been tried in gonorrhœa, but he did not think it was of any great value. Mr. Wyndham Powell had referred to the abortive treatment by nitrate of silver, and he (Mr. Ryall) certainly thought it was the method which should be adopted in early stages, *i. e.* wipe out the urethra with nitrate of silver. He agreed with Mr. Powell as to the height of the irrigator. His own remarks applied to irrigation of the whole of the lower urethra and urinary tract, not to anterior irrigation alone. His experience was that where very high pressure was used, as advocated by Valentine, it was impossible to irrigate the posterior urethra unless cocaine were used.

The speakers were cordially thanked by the members for their contributions.

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# THE CLINICAL JOURNAL,

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# ON THE DIAGNOSIS OF MEDICAL DISEASES OF JOINTS.*


By W. HALE WHITE, M.D., F.R.C.P.,
Physician to Guy's Hospital.


GENTLEMEN,—When you kindly asked me to read before you a paper on medical diseases of joints, I gladly accepted, for it is a subject in which I have for many years been interested, and my teaching at the bedside about some cases has been different from that in many text-books, but it is a great satisfaction to find that this teaching is gradually becoming accepted as probably correct, as we may see by consulting the last volume of Clifford Allbutt and Rolleston's 'System of Medicine,' issued a few months ago, where the whole subject is dealt with by Dr. Garrod.

I propose this evening to deal chiefly with difficulties of diagnosis. The first point about which I would try to convince you is that from among the diseases named indifferently "osteo-arthritis," "rheumatoid arthritis," "chronic articular rheumatism," or "arthritis deformans," we can pick out a number of cases sufficiently alike clinically and sufficiently distinct from the rest to be regarded as a separate disease, the clinical course of which is such that the cause of the disease is almost certainly bacterial; and it may be that in years to come we shall be able to further sub-divide these cases, for perhaps in some the condition is caused by one form of bacteria, in others by another. To this disease the name "rheumatoid arthritis" should be restricted, and is coming to be so restricted. If much pyrexia is present it may be called "acute rheumatoid arthritis," if the acute stage has passed off and left the joints deformed it is called "chronic rheumatoid arthritis." The advantage of this nomenclature is that no new name is introduced; the disease is certainly an arthritis, and as

* Delivered before the Hampstead Medical Society.

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when it is acute it has been confused with rheumatic fever, rheumatoid arthritis is a good name.

The patient afflicted with rheumatoid arthritis is usually a woman somewhere between the ages of twenty and forty. When seen in her first attack there is usually some pyrexia, but not much. The temperature is raised to perhaps 100° or 101° F. in the evening and is lower in the morning. The pyrexia lasts a few weeks and then slowly subsides. The temperature charts I pass round show this. Sometimes the temperature is raised more, sometimes less than this. The rate of the pulse is much higher than the temperature would lead us to expect; it is often over 100 and may be 140. It is much the same night and morning, and remains rapid even after the temperature has fallen. This rapidity of pulse is a very important and constant feature, and is not associated with any evidence of valvular disease of the heart. A number of joints are affected in quick succession; pain is an early symptom of the articular affection. Usually the patient is not seen by a doctor until she has been ill some days and many joints are swollen. Those between the first and second phalanges of the fingers are affected earliest, and by the time the patient is seen spindle-shaped swelling around these joints is very evident. I pass round photographs illustrating this. The fact that the swelling is spindle shaped shows that it extends beyond the joint itself to the soft tissues near it. It is particularly to be observed that the terminal phalangeal joints are hardly ever affected, and in this respect the disease forms a striking contrast to osteo-arthritis, in which the affection of the terminal joints as shown by the presence of Heberden's nodes is so common. The metacarpo-phalangeal joints are also implicated. The two hands are always symmetrically affected, and often the inter-phalangeal joints between the first and second phalanges of the toes are the seat of spindle-shaped swellings, and there is hardly anything more characteristic in clinical medicine than this spindle swelling of these interphalangeal joints. The metacarpo-phalangeal joint of the thumbs, the wrists, ankles, knees, elbows, hips and shoulders may all be affected, and when the anatomical disposition of the parts allows it the spindle shape of the swelling is evident. The trouble is a painful articular and peri-articular inflammation. This disease differs from those maladies with which it is likely to be confused in the fact that the temporo-maxillary joints and those of the spine are usually and severely implicated. The patient is pale, lies in bed unable to move, except with difficulty, not only because of the immobility of the joints, but also because of the great pain. The hands and feet are often found to be sweating profusely, and usually the arthritic muscular atrophy is very severe, more so perhaps than in any other form of arthritis, and hence after the patient has been ill a little while she is very wasted. On carefully examining her pigmentation may often be seen, but it is not so constant as the other signs. The most common variety consists of a number of freckles, which appear chiefly on the limbs. They are of varying shades of brown; the less common variety consists of a brown pigmentation of the face. Both varieties often slowly disappear as the acute symptoms pass off. It is not unusual to find the various lymphatic glands of the body a little enlarged.

Let us return to the joints. Examination during life, both tactile and by the X rays, shows that the disease is not one of the bones. I show you some X-ray pictures which illustrate that no bony outgrowths or alterations in the bones occur. I am fortunate enough to have had under my care a woman who, while suffering from this disease, died of typhoid fever. I show you a section of one of her phalangeal joints, and you will see that there is considerable inflammation of the synovial membrane. The disease is fundamentally an inflammation of the synovial membrane, and the soft tissues around the joint, hence the uniform spindle-shaped swelling. The fringes of the synovial membrane hypertrophy, and if one of these fringes presses upon the cartilage it becomes eroded and the bone is exposed, and even it after a long while may be mechanically irritated by some such fringe, and thus some of the bone may be eroded and a little bony hypertrophy may take place around the erosion. But this implication of cartilage and bone is quite accidental; it is often absent. Usually the bone and cartilage are quite healthy. This is most important, as the limitation of the disease to the synovial membrane is a fundamental difference between this disease and osteo-arthritis. The X rays show a considerable translucency of the bone in the neighbourhood of the joints. This is seen in other varieties of arthritis, *e. g.*

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gonorrhœal, but it is especially striking in rheumatoid arthritis.

Slowly the acute symptoms subside. In an average case the temperature is normal at the end of a month, the pain lessens and the swelling about the joints decreases; the anæmia, which is usually present during the acute stage, improves, and the patient feels better. But she is not well, for some swelling remains about the joints, some pain persists, movement is limited, a good deal of muscle atrophy remains, and the pulse continues above the normal rate. Few patients completely recover from an acute attack; after it they remain to some extent invalided. Sooner or later another acute attack comes on, often in a few months. It exactly repeats the first, except that it is probably a little less severe, but after the second the permanent damage to the joints is greater; and so the disease progresses—acute or subacute attacks follow one another at varying intervals. Each attack is for the most part less severe than the one that preceded it, and finally the disease wears itself out, but the patient is a chronic invalid who suffers from more or less constant pain in her joints, some deformity of them, wasting of the muscles, a rapid pulse, and in a few cases pigmentation of the skin. Many of the patients who flock to Bath, Aix, and other places famed for the treatment of rheumatic affections, are instances of the chronic stage of rheumatoid arthritis. They are often confused with sufferers from osteo-arthritis, but the distinctions are that the sufferers from the chronic stage of rheumatoid arthritis are, as a rule, younger; most of the joints in the limbs and body are affected, including the jaw, but the terminal phalangeal joints are spared. Heberden's nodes are never present; the joints implicated, especially the phalangeal, have a spindle swelling around them, and there are rarely bony out-growths, and when present they are inconspicuous. The above description applies to a case of average severity; some are slighter, some much more severe; in these the patient may be bedridden. It should be added that sometimes the affection of the temporo-maxillary joint leads to difficulty of swallowing. The cause of this disease has not been made out. The attempt has often been made to isolate a specific micro-organism, but so far without success.

There is a multiple affection of joints occurring in young children and associated with enlargement of the lymphatic glands, which was first described by Dr. Still, and is called after him. In these cases the essential change lies in the synovial membrane and the tissues around the joint, and most writers now think that these are only examples of rheumatoid arthritis occurring in young children.

This disease—rheumatoid arthritis—is not rare. The diagnosis may be frequently seen over the beds of patients in the hospital, and I feel sure that if I could have here a patient to show you I could convince you that such a disease exists. As proof of this I may mention that since some years ago, when first I began to be aware of the existence of the disease, no house-physician who has worked with me has finished his term of office without being convinced of the reality of the disease. When we have admitted patients in the acute stage they have often been sent in with the diagnosis of rheumatic fever, but when rheumatoid arthritis is recognised as a definite disease the distinction is easy, for in rheumatic fever the arthritis soon leaves one joint and appears in another. The phalangeal joints and those of the spine and jaw are rarely affected in rheumatic fever; the temperature falls more quickly in rheumatic fever; the sweating is all over the body and is not confined to the hands and feet; pigmentation is not common in rheumatic fever, nor is wasting extreme. Cardiac trouble is common in rheumatic fever and very rare in rheumatoid arthritis; great rapidity of pulse apart from cardiac disease is not common in rheumatic fever, and rheumatic fever does not cause a spindle-shaped swelling of joints, nor leave behind a permanent defect of joints except in very rare cases. I can, however, give you an instance of this. A boy, æt. 12 years, was admitted under my care. He had been in the hospital at the age of 5 with chorea, at the age of 7 with pains and swelling of several joints, endocarditis and pyrexia, at the age of 8 with erythema, fresh endocarditis, pain and swelling of several joints relieved by salicylates. When admitted at the age of 12 there was much muscular atrophy, with fixation of all the joints of all four limbs in a position of flexion; the movements of the neck and jaw were impaired. It is true that some of his lymphatic glands were enlarged, but the chorea, erythema and endocarditis made us regard his joint trouble as rheumatic. The glands which were chiefly enlarged were those

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of the neck, and their enlargement could be explained by chronic eczema of the scalp. He died eight years later at his own house. My house-physician, Dr. H. J. Starling, made a post-mortem examination. The lungs were riddled with phthisical cavities, the pericardium was adherent, and there were vegetations on the mitral valve. The evidence in this case seems so strong that we must allow that the patient had chronic joint disease following acute rheumatism, but when we remember the great frequency of acute rheumatism it is clear that chronic joint disease following it is excessively rare, and I would strongly urge that chronic disease of joints should never be ascribed to acute rheumatism, unless the evidence of previous acute rheumatism is very strong.

It has been suggested that rheumatoid arthritis is really a chronic infective arthritis. This may well be so, but if it is we do not know the seat of infection, nor the invading micro-organism. It is a golden rule in chronic diseases of joints, as in acute, to look out for a seat of infection. Not long ago a man was in the hospital with chronic arthritis affecting, and hopelessly deforming and fixing, most of the joints in his body. Pus was oozing up around his teeth, and if the condition, which was no doubt a chronic pyæmic arthritis, had been recognised earlier, his being a cripple might have been prevented, for had the teeth been properly attended to the joint trouble would have subsided. But I am quite sure from having looked at many cases that usually, if the diagnosis of rheumatoid arthritis is made on sufficient clinical grounds, no source of pyæmic infection will be found.

Before leaving this subject I should like to remind you of the importance of looking to the urine in cases of diseases of the joints. Not long ago I saw an old lady who was thought to have chronic articular rheumatism, but on examination it was found that she had pyuria, and then we found out that the joints had only been affected since the pyuria began, and that those implicated were the shoulders, ankles, and right knee. The shoulders were nearly fixed and there was grating in the right; the ankles and knees were swollen and tender. There was no affection of the hands and feet as is so common in chronic articular rheumatism. Again, a few days ago, I saw a lady who had been thought to have gout, for she had pains in her oints, and when first given colchicum relieved the pain; but later on the trouble became much worse, pus was found in her urine, and also a pure culture of *Bacillus coli*, and it must be remembered that the urine may swarm with virulent bacteria and yet contain no albumen. We are treating this patient successfully with a vaccine obtained from her own bacilli.

The question has been raised whether some cases which at first sight appear to be rheumatoid arthritis are not really examples of tubercular disease of joints. Usually speaking, tubercular disease of joints is quite unlike rheumatoid arthritis and quite easy to diagnose. I feel sure that the majority are not, for no tubercular lesions can be detected elsewhere. I have not had a case in the hospital since the introduction of Calmette's reaction, but probably that would help us considerably.

Speaking of tubercular arthritis reminds me that it may also be confused with osteo-arthritis. To give you an example of the difficulty, a woman, æt. 40 years, had phthisis with tubercle bacilli in the sputum; her elbows, ankles, shoulders, and wrists were affected with a disease which would, if she had been otherwise healthy, have certainly been regarded as rheumatoid arthritis. Another instance was that of a man who had fixation of the left hip. There was some rheumatic change in two phalangeal joints of the fingers and one of the toes. Some thought the hip tubercular, others were sure it was not, but a tubercular abscess ultimately developed in connection with it.

The words "osteo-arthritis" and "rheumatoid arthritis" are often used loosely and as though they were interchangeable, but this should never be done. We have seen that rheumatoid arthritis is a distinct disease both clinically and in the post-mortem room. In osteo-arthritis the only variety that could in any way be confused with rheumatoid arthritis is that in which many joints are affected early in the disease, but even then the distinction is not usually difficult. We do not in osteo-arthritis get the spindle-shaped swellings on the proximal phalangeal joints, but we do find Heberden's nodes on the terminal phalangeal joints. Quite early in the course of osteo-arthritis we can obtain clinical evidence of destruction of cartilage, and soon also of bony outgrowth, but we do not find the same degree of swelling around the joints as we see in rheumatoid arthritis which occurs at a

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younger age, is accompanied by a rapid pulse, by sweating of the hands and feet, pigmentation of the skin, and extreme muscular atrophy. It is hardly possible for mistakes to be made between the usual form of osteo-arthritis and rheumatoid arthritis, for the former commonly attacks the knee or hip by preference, and is usually confined to one or two joints for a long while.

Passing on now to the conditions with which osteo-arthritis can be confused, first I should like to mention the pads described by Dr. Garrod and often seen on the dorsal aspect of the proximal phalangeal joints of the hands. These are quite common, but I believe have nothing to do with osteo-arthritis or gout, to both of which they are frequently ascribed. They are not infrequently associated with Dupuytren's contraction of the palmar fascia, and the origin of both is equally obscure. They are quite unconnected with the joints or the bones, and I pass you round some X-ray photographs which demonstrate this very clearly. The other day I was fortunate in seeing a patient who had them. The surgeon who saw him decided to excise the palmar fascia, and the patient permitted one of these pads to be cut off. Sections of it showed it to be composed of fibrous tissue with normal skin over it.

Very often patients come before us and assure us they have gout. I really think that more patients ascribe their Heberden's nodes to gout than to osteo-arthritis. We do know definitely that after death urate of soda is found deposited in gouty joints, and I would plead that no patient should be said to have gout unless urate of soda can be seen, or the clinical course of his trouble is so like that of cases in which we know urate of soda to be present that there is a strong probability that we may correctly assume urate of soda to be present in his joints. It may be quite impossible to say whether a particular patient is suffering from osteo-arthritis or gout, but that is no justification for calling his disease rheumatic gout.

Chronic osteo-arthritis of the hip is very common, and so is sciatica, and as both cause pain in or near the hip mistakes are often made; it would be out of place that I should here spend time on the points of distinction; the mistake can usually be avoided if sufficient care is taken. A few months ago I saw a case in which many members of our profession, even after taking great care, had been led astray, for a case of sarcoma of the head of the tibia was for a long time thought to be one of osteo-arthritis of the knee. In this particular case I hardly know how, in the early stages, the mistake could have been avoided.

Although usually the joint troubles that occur in nervous diseases are easily diagnosed, yet mistakes are made. It is a good thing to examine the knee-jerks and pupillary reflex, for the first symptom of tabes may be disease of the joints, and although the distinction between a typical osteo-arthritic joint and one typical of tabes is, in most cases, quite easy, yet in a few cases it is hard to tell one from the other. The disorders of joints that occur in the rarer nervous diseases are more often overlooked; thus I have known a joint affection due to syringomyelia scraped, the syringomyelia having been quite overlooked. One of the most curious I have myself encountered was that of a man who was sent in for supposed rheumatic fever. The lower extremities were very tender, and he said he could not move them. The left knee was swollen, but as the skin was very dry I doubted the diagnosis. Two days after admission the right ankle became swollen and red. Two days after an acute trophic bed-sore developed; he had cystitis, but the patient ultimately recovered from a severe attack of acute myelitis, which was the cause of his arthritic trouble.

There are other rare diseases about which mistakes are occasionally made. Perhaps the most disastrous is to fail to recognise the arthritis which may accompany hæmophilia, for surgical interference with the joint may cost the patient his life. Children with Henoch's purpura often complain of pains in the joints, and I have known a child suffering from Henoch's purpura which was mistaken for chronic rheumatism.

From time to time we all come across cases in which gonorrhœal rheumatism is overlooked. I have known it called rheumatic fever; it may be confused with rheumatoid arthritis. It sometimes leads to a severe degree of muscular wasting, and twice I have known cases which were really examples of gonorrhœal rheumatism set down to progressive muscular atrophy. In countries in which Malta fever and gonorrhœa are both common, as arthritis accompanies both diseases mistakes are liable to occur. The best way to avoid error is always to have in mind the possibility of gonorrhœal rheumatism. I have seen several examples in women.

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There is no necessity for the infection to take place in the genital organs; many cases have occurred in which gonorrhœal rheumatism has followed infection of the eye, and thus it may in young infants follow purulent inflammation of the conjunctiva. Sometimes the diagnosis is very difficult, for gonorrhœal rheumatism may follow many weeks after the gonorrhœa is apparently cured. In such a case it has not been completely cured; some gonorrhœal inflammation of the prostatic urethra remains, and from this the gonorrhœal rheumatism is derived. The chief distinctions from rheumatoid arthritis are that in gonorrhœal rheumatism, the knees are most often affected, and we rarely see the spindle-shaped swelling of many fingers, but the sheaths of tendons and bursæ are often affected; also there is commonly a pain at the back part of the sole of the foot. Rheumatoid arthritis is far commoner in women than in men, and is accompanied by a rapid pulse, sweating of the hands and feet, and pigmentation. Rheumatic fever is such a well-known disease that I need hardly discuss the distinctions between it and gonorrhœal rheumatism, but as we saw a moment ago the mistake is made, and usually from want of care.

But many diseases other than gonorrhœal rheumatism and rheumatoid arthritis are mistaken for rheumatic fever. The percentage of errors in connection with rheumatic fever is not high, but the total is, for rheumatic fever is so common. Usually the diagnosis is so easy that we diagnose, say, forty-nine cases successfully, and then when the fiftieth difficult case meets us we overlook it.

There is a group of errors in which the mistake arose from diagnosing rheumatic fever, although only one joint was implicated. I believe the diagnosis of rheumatic fever should always be withheld so long as only one joint is affected. Early in the disease a single joint may be inflamed, but while this is so leave the diagnosis in doubt. If the patient is suffering from rheumatic fever a second joint will be affected within twenty-four hours. I have twice known fracture into the knee-joint mistaken for rheumatic fever. I have seen the same error made in a case of osteo myelitis of the head of the tibia, and this reminds me of a case which hardly illustrates the point under discussion, but is worthy of mention, for the patient had osteo-myelitis of the head of each tibia, but rheumatic fever was diagnosed. In a young girl acute necrosis of the head of the femur with effusion of pus into the hip-joint was regarded as rheumatic fever. In all these cases the error was soon recognised, and the patient was handed over to a surgeon, but I once saw a child who was suddenly taken ill with pain in the hip and fever. He was thought to be suffering from rheumatic fever and was admitted into the hospital five days after the onset of acute symptoms. Acute necrosis of the head of the femur was diagnosed. The hip-joint was at once opened; the diagnosis was confirmed, but the patient died from septicæmia a few hours after. On several occasions I have known pleurisy or pneumonia to cause such pain in the shoulder on the same side as the pulmonary disease that the patient has been thought, as pyrexia was present, to be suffering from rheumatic fever. It is true that usually the pleurisy or pneumonia has been discovered, but the case has been wrongly regarded as rheumatic fever with rheumatic pleurisy instead of, as it ought to have been, pneumococcal pleurisy or pneumonia with pain in the shoulder. Sometimes I have known pleurisy or pneumonia to be completely missed because attention has been so concentrated on the pain in the shoulder, which was thought to be rheumatic. In these cases pain is not due to pneumococcal affection of the joint, for no effusion ultimately occurs. I might give many other instances from my own personal knowledge in which refusal to diagnose rheumatic fever when only one joint was affected has saved a mistake.

Rheumatic fever may be mistaken for almost any specific fever, because the patient who has fever often complains of pain in the limbs, and if these pains are referred to the joints an erroneous diagnosis may be made. Perhaps the mistake is commonest with influenza, but I have even heard of smallpox being mistaken for rheumatic fever, but in most cases the lapse of a few days sets the diagnosis right. This is often not so when pyæmia is mistaken for rheumatic fever. Such an error is by no means uncommon, and so important that it is worth while to mention a few examples. Some years ago a youth was transferred to my ward from a surgical ward where pain and effusion in several joints had led to a diagnosis of rheumatic fever. A rectal examination revealed a prostatic abscess, which was the source of the pyæmia. On another occasion a patient was sent in as a case of rheumatic fever, but a careful examination showed him to be suffering from pyæmia due to a sore on the heel. In another case rheumatic fever was incorrectly diagnosed in a patient suffering from pyæmia due to suppuration of both middle ears. In another I remember the patient was sent in as a case of rheumatic fever, but the case was one of pyæmia following a cut on the hand. Pus was ultimately let out of the shoulder-joint. The con-

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fusion between rheumatic fever and pyæmia is very serious, for if pyæmia is called rheumatic fever, failure to adopt surgical treatment may cause the patient to lose his life, and an almost certain hope of recovery may be held out when he is within a few days of his death. If pyæmia takes the form of malignant endocarditis the diagnosis may be difficult, as in the case of a boy who was admitted under me. He was one of a large family, several of whom had had rheumatic fever and chorea. He had much pain and some swelling of both knees, his temperature was 104·4° F., and he had systolic and presystolic mitral murmurs. Rheumatic fever was thought to be possible, but the diagnosis was regarded as doubtful as the skin was so dry. In forty-eight hours signs of meningitis showed themselves, and a diagnosis of malignant endocarditis with pyæmic infection of the meninges and knees was made. He died, and this diagnosis was correct: there was pus in both knees. More than once I have known syphilis, which has caused effusion into the joints, to be mistaken for rheumatic fever. The following is an example of this error. A man, æt. 26 years, was sent into the hospital with a diagnosis of rheumatic fever, which he had had twelve years before. The present illness began three weeks before admission, with pain and swelling of the left elbow. A week later the left knee was affected in the same way, and soon after the left foot. On admission he had pyrexia, the above-mentioned joints were swollen, and he was sweating considerably; there was slight, but distinct, evidence of a secondary syphilitic rash; he had a sore throat, and had had a hard chancre ten weeks ago. Forty-eight hours' treatment by salicylates made no impression on the disease, but he finally recovered with mercury. I have known cerebro-spinal meningitis to be mistaken for rheumatic fever. Pyrexia with pain and effusion into the joints are early symptoms in this form of meningitis; the rash may be thought to be a rheumatic erythema, and the pain caused by retraction of the head may be set down to rheumatism.

In countries where dysentery is common, the arthritis which sometimes accompanies may give rise to difficulty, as may that which in this country is associated with glanders. The febrile pains which are often an early symptom of typhoid have led to errors, but the arthritis of this disease usually does not come on till such a time that the diagnosis of typhoid has been made, and therefore ought not to give rise to mistakes. Pneumococcal arthritis has been thought to be rheumatic, and arthritis may accompany any febrile disease. I have not discussed the question of scarlatinal rheumatism, as it cannot be said to be definitely settled for all cases whether the arthritic symptoms are not due to genuine rheumatic fever occurring as a complication of scarlet fever.

*May 4th, 1908.*

# A REVIEW OF MORTALITY STATISTICS DURING THE LAST HALF CENTURY.*

By SIDNEY PHILLIPS, M.D., F.R.C.P.
Physician to St. Mary's Hospital.

GENTLEMEN,—The laws of the Society require from the President an address on some subject of interest, and I hope that a short survey of the effects of disease, as shown by the mortality returns of the Registrar-General during the last half century, may come under that head. I take the year 1850 as the earliest year to look back to, for it was from this date that the issue was commenced of the decennial reports, which are of such service in availing ourselves of the labours of successive superintendents of statistics to the Registrar-Generals, not the least distinguished of whom is the present holder of the office, Dr. Tatham. The last decennial report ended with the year 1890, but I have compiled tables on the same lines as those in the decennial reports, bringing the figures up to the end of 1905, the latest year for which an annual report has been published.

It must be remembered that before 1875, notification not being compulsory, omissions in registration may have occurred, and that any decrease in mortality since that date is, therefore, under- rather than over-stated.

I have drawn up the annexed table to show in round numbers the disease distribution of deaths in 1855, side by side with that in 1905:

*Disease Distribution of each Thousand Deaths.*

| | 1855. | 1905. |
|---|---|---|
| Diseases of respiratory organs | 256 | 250 |
| ,, ,, circulatory ,, | 60 † | 150 ‡ |
| ,, ,, abdominal ,, | 70 | 100 |
| ,, ,, nervous system | 140 † | 80 ‡ |
| ,, ,, urinary ,, | 16 | 30 |
| Exanthemata | 170 | 75 |
| Influenza | 0 | 13 |
| Cholera | 3 | 0 |
| Malignant disease | 17 | 60 |
| Puerperal ,, | ? | 12 |
| Rheumatism and gout | ? | 8 |

* Presidential Address delivered before the Harveian Society.

† Excluding apoplexy. ‡ Including apoplexy.

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| | 1855. | 1905. |
|---|---|---|
| Diabetes | 2 | 7 |
| Alcoholism | ? | 4 |
| Rickets, alcoholism, atrophy | ? | 95 |
| Violence | 34 | 36 |
| Old age | ? | 60 |
| Unclassified | 232 | 20 |
| Total | 1000 | 1000 |

*Diseases of the Respiratory System.*

These account for no less than one quarter of the deaths from all causes.

Of the 250 deaths in each 1000:

| | |
|---|---|
| Bronchitis accounted for | 75 deaths |
| Pneumonia " " | 85 " |
| Phthisis " " | 75 " |
| Other diseases (asthma, pleurisy, emphysema, etc.) | 15 " |
| Total | 250 |

*Bronchitis*

is, in many cases, only a secondary cause of death, but it finds a separate heading in the Registrar's returns; from these it appears its frequency is greatly decreasing.

*Deaths Annually per Million Living from Bronchitis.*

| Decennium. | Deaths. |
|---|---|
| 1851–1860 | no return |
| 1861–1870 | 1691 |
| 1871–1880 | 2186 |
| 1881–1890 | 2081 |
| 1891–1900 | 1811 |
| 1901–1905 (5 years) | 1220 |

As the Registrar points out, probably one reason for the decline is that many deaths formerly certified as from bronchitis are now more accurately certified as from broncho-pneumonia.

*Pneumonia.*

In the nomenclature of the Royal College of Physicians pneumonia was classed with local diseases until 1900, when it was transferred to the group of infective diseases. And since 1900 the Registrar-General's returns subdivide pneumonia into lobar pneumonia, broncho-pneumonia, epidemic pneumonia, and cases of which the variety is not defined. These divisions are often difficult to make, as appears from the fact that in about half the cases medical men decline to certify more than that death occurred from pneumonia; and the term "epidemic pneumonia" seems so little accepted that during the years 1903, 1904, and 1905, only eleven cases altogether were so certified—little enough for an epidemic disease. Disregarding these subdivisions and taking all cases of pneumonia together, there is an increase of 17 per cent. in the deaths from pneumonia in the last forty-five years.

*Deaths Annually from Pneumonia per Million Living.*

| Decennium. | Deaths. |
|---|---|
| 1851–1860 | No return |
| 1861–1870 | 1089 |
| 1871–1880 | 1004 |
| 1881–1890 | 1066 |
| 1891–1900 | 1227 |
| 1901–1905 (5 years) | 1277 |

As already pointed out, some part of this rise may be due to deaths which would formerly have been certified as from bronchitis being now certified as from pneumonia.

Professor Osler, in an address on "Pneumococcal Infections" at the Medical Society of London (December 9th, 1907), points out that "among the infective diseases of modern life, pneumonia infection alone is on the increase." In Chicago the mortality reached as high as 2000 per million inhabitants, chiefly affecting the coloured population.

In this country, however, the increased mortality from influenza has been almost coincident with the prevalence of influenza which commenced to be epidemic in December, 1889.

The following table shows the pneumonia mortality in each of the ten years immediately *preceding* the influenza epidemic:

*Deaths from Pneumonia per Million Living.*

| Year. | Deaths. | Year. | Deaths. |
|---|---|---|---|
| 1880 | 976 | 1885 | 1074 |
| 1881 | 910 | 1886 | 1021 |
| 1882 | 989 | 1887 | 1113 |
| 1883 | 1059 | 1888 | 1093 |
| 1884 | 972 | 1889 | 1022 |

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These figures show little increase in pneumonia mortality before influenza appeared.

The following table shows the pneumonia mortality in each year since influenza began, side by side with the influenza mortality:

*Deaths from Influenza and Deaths from Pneumonia per Million Living.*

| Year. | Deaths from influenza. | Deaths from pneumonia. |
|---|---|---|
| 1889 | 2 | 1022 |
| 1890 | 157 | 1404 |
| 1891 | 574 | 1471 |
| 1892 | 534 | 1250 |
| 1893 | 325 | 1285 |
| 1894 | 220 | 1082 |
| 1895 | 423 | 1169 |
| 1896 | 122 | 1146 |
| 1897 | 196 | 1118 |
| 1898 | 330 | 1125 |
| 1899 | 389 | 1250 |
| 1900 | 500 | 1374 |
| 1901 | 174 | 1147 |
| 1902 | 223 | 1437 |
| 1903 | 189 | 1220 |
| 1904 | 168 | 1281 |
| 1905 | 204 | 1299 |

Possibly owing to the varying forms in which influenza makes its attacks, the influenza and pneumonia mortalities do not invariably correspond, but it will be seen that when influenza began the pneumonia mortality suddenly rose from 1022 per million living in 1889, to 1404 in 1890, and in 1891 and 1900, the two years of highest mortality from influenza, the pneumonia mortality was almost at its maximum, reaching 1471 per million in 1891—the highest ever recorded.

It appears, therefore, that in England the increased mortality from pneumonia (apart from other pneumococcal affections), is in great part, at any rate, accounted for by the prevalence of influenza since 1889.

(P.S.—The figures for 1906 confirm this conclusion, the influenza mortality falling to 183, while the pneumonia mortality fell to 1210 per million living.)

### *Phthisis.*

*Deaths Annually from Phthisis per Million Living.*

| | |
|---|---|
| In the decennium 1851–1860 | 2730 |
| „ „ 1861–1870 | 2545 |
| In the decennium 1871–1880 | 2190 |
| „ „ 1881–1890 | 1775 |
| „ „ 1891–1900 | 1391 |
| In the five years 1901–1905 | 1215 |

The Registrar-General points out that certification and registration of deaths have undergone many changes of late years, and too much stress must not be laid on the declining death-rate from phthisis.

But the decline is steady and continuous, and while the mortality from all forms of tuberculosis taken together has decreased by 42 per cent., that from pulmonary tuberculosis has fallen most of all, viz. by 55·5 per cent.; the importance of this may be gauged when it is remembered that of every 100 deaths from tuberculosis 69 are from pulmonary tubercle.

The lessened mortality from phthisis cannot be wholly attributed to sanatorium treatment, for the decline in the death-rate was almost, if not quite, as marked before the time when sanatorium treatment was taken up.

### *The Remaining Diseases of the Respiratory Organs.*

Asthma, pleurisy, emphysema, laryngitis, together produced only 15 of the 250 deaths per thousand arising from respiratory diseases, and there appears to be no material alteration in their fatality in the last fifty years.

*Deaths Annually per Million Living from Pleurisy and from Laryngitis.*

| Decennium. | Deaths from pleurisy. | Deaths from laryngitis. |
|---|---|---|
| 1861–1870 | 42 | 47 |
| 1871–1880 | 50 | 45 |
| 1881–1890 | 56 | 52 |
| 1891–1900 | 54 | 45 |
| 1901–1905 (5 years) | 36 | 33 |

The decrease in "laryngitis" is probably due to laryngitis being now often certified under "diphtheria."

### *Diseases of the Circulatory System*

caused in the year 1905 about 150 of each 1000 deaths. Of these about 93 arose from cardiac disease and 57 from diseases of the blood-vessels.

### *Cardiac Diseases.*

The 93 per 1000 deaths from heart disease included deaths from hypertrophy, dilatation, and

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fatty and valvular disease of heart, and from angina pectoris; these conditions are separately enumerated in the death returns (and since 1900 also infective endocarditis), but they occur in combination so constantly that their attempted separation is valueless statistically, and I have massed together the various conditions in the following table:

*Deaths Annually per Million Living from Cardiac Diseases.*

| Period of time. | Deaths. |
|---|---|
| 1851–1860 (10 years) | 1160 |
| 1861–1870 ,, | 1054 |
| 1871–1875 (5 years) | 1217 |
| 1876–1880 ,, | 1415 |
| 1881–1885 ,, | 1259 |
| 1886–1890 ,, | 1640 |
| 1891–1895 ,, | 1553 |
| 1896–1900 ,, | 1515 |
| 1901–1905 ,, | 1409 |

From this table there appears to be no material alteration in the fatality from heart disease.

*Diseases of the Blood-vessels*

accounted for 55 of each 1000 deaths in 1905. No less than 45 of these were from "apoplexy," cerebral hæmorrhage or embolism; till the year 1900 "apoplexy" was classed among diseases of the nervous system, but since then has been transferred to the group of diseases of the arteries. During the last few years the deaths certified from apoplexy have much lessened in number.

Many deaths from apoplexy are now certified under the head of nephritis, which was the primary cause of the arterial disease that led to the hæmorrhage; this may in part account for the decreased certificates of deaths from apoplexy.

Increased recognition has been given of late years to affections of arteries, and the deaths certified from arterial disease are increasing in consequence.

(P.S.—The report for 1906 shows a further increase to 871 per million living.)

*Abdominal Diseases.*

I include in this group diseases of the alimentary canal, liver, and gall-bladder, together with tubercular abdominal affections and the group of affections figuring in the Registrar's returns as "diarrhœal diseases." Abdominal disorders accounted for about 100 of each 1000 deaths in 1905.

The proportion of deaths from individual abdominal affections cannot be treated of here, but I select some for comment which are of special interest.

The 100 deaths thus made up:

| | |
|---|---|
| Tubercular abdominal disease | 10 |
| "Diarrhœal diseases" | 40 |
| Appendix disease | 4 |
| Gastric ulcer | 3 |
| Cirrhosis of liver | 8 |
| Other abdominal diseases | 35 |
| | 100 |

*Tubercular Abdominal Disease*

includes tubercle of peritoneum, intestines and mesenteric glands, described formerly by the now discarded term "tabes mesenterica."

*Annual Mortality per Million Living from Abdominal Tuberculosis.*

| | | | |
|---|---|---|---|
| In the decennium | 1861–1870 | 266 | deaths. |
| ,, ,, | 1871–1880 | 285 | ,, |
| ,, ,, | 1881–1890 | 257 | ,, |
| ,, ,, | 1891–1900 | 207 | ,, |
| ,, 5 years | 1901–1905 | 166 | ,, |

The table shows that in common with tubercular disease in other parts of the body abdominal tubercle has greatly diminished in fatality — a decrease of 37 per cent. in 45 years, much the same as the decline in deaths from tubercle of the meninges, but less than that from phthisis. The deaths are mainly among infants under five years of age.

*Diarrhœal Diseases.*

Before 1869 typhoid fever was not differentiated from other febrile conditions, and many cases of typhoid were probably included in the group of "diarrhœal diseases"; previous to the year 1874, cholera nostras, cholera infantum, Asiatic cholera, and dysentery were returned under the head of "diarrhœa," and as recently as 1901 infantile cholera, cholera nostras and choleraic diarrhœa were included in the tables under "diarrhœal diseases." The last edition of the 'Nomenclature of Diseases of the Royal College of Physicians' directs that the term "diarrhœa" is only to be used in death certificates when its cause is unknown, and the term "epidemic diarrhœa" or "infective

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enteritis" is to be employed in other cases, and further, whenever diarrhœa is due to food-poisoning the fact should be recorded: this is often impossible, and only 211 cases were certified as resulting from food in 1905.

Many practitioners still continue the use of the terms "gastro-enteritis" or "gastro-enteric catarrh" in death certificates, and these terms appear to correctly describe the causes of many fatal diarrhœas.

*Deaths Annually from Diarrhœa per Million Living.*

| Decennium. | Deaths. |
|---|---|
| 1871–1880 | 843 |
| 1881–1890 | 631 |
| 1891–1900 | 738 |
| 1901–1905 | 671 |

The greater number of the deaths are those of young infants. No doubt improper feeding is accountable for many of these deaths, but there are very wide variations in individual years, largely due to climatic conditions, a hot summer increasing the diarrhœal mortality and the converse. The high rate in 1891–1900 was due to the series of unusually hot summers of 1892, 1893, and 1894. (P.S.—The Report for 1906, since published, shows a mortality of 886 per million living in consequence of the hot summer in that year.) The effect of a cold summer is illustrated in the Registrar's report, just published, of the mortality in the third quarter of 1907: "The death-rate—12·1—was 3·9 below the mean in the ten preceding third quarters, and is the lowest recorded in any quarter since civil registration began; the abnormal fall in the death-rate was mainly due to the showery and exceedingly cool summer, which checked the rise in the rate of child mortality that usually occurs in this quarter." In hot summers the difficulty in obtaining and in preserving milk adds to the diarrhœal mortality, but this might, in my opinion, be lessened were opium more freely used in its treatment: there is a tradition that infants take opium only with danger, but I find, on the contrary, that they take it very well, and I believe it is the only effective treatment in summer diarrhœa of infants.

*Appendicitis.*

The death returns afford little assistance in ascertaining if appendicitis has become more frequent of late years. Before the year 1901 deaths from appendicitis were included in the group of "peritonitis, enteritis, and typhlitis." The number of the deaths in this group had been for many years previous to 1901 steadily decreasing, and so far as death returns go they do not support the view that appendicitis was becoming more fatal before 1901.

Since 1901 appendicitis has had a separate heading in the death returns:

| | | Per million living persons. |
|---|---|---|
| In 1901 appendicitis caused | 1244 deaths | = 38 |
| ,, 1902 ,, ,, | 1485 ,, | = 45 |
| ,, 1903 ,, ,, | 1729 ,, | = 52 |
| ,, 1904 ,, ,, | 1887 ,, | = 56 |
| ,, 1905 ,, ,, | 1946 ,, | = 57 |

From this it would, at first sight, appear that appendicitis is becoming more fatal. But the deaths from peritonitis have decreased in exactly the same numbers as the deaths from appendicitis have increased in these five years; thus in 1901 the deaths from peritonitis were 1532, and from appendicitis were 1244, a total of 2776; in 1905 the total was almost exactly the same—2789, but of this total only 843 deaths were certified as from peritonitis, and no less than 1946 from appendicitis.

It is presumable, therefore, that the seeming increase in deaths from appendicitis is due to more accurate certification.

Of course the death-rate is not necessarily a measure of the *prevalence* of the disease, but so far as it goes it does not show any real increase in the mortality from appendicitis. (P.S.—In 1906 the deaths rose still further to 62 per million.)

*Gastric Ulcer*

was not tabulated separately in the returns till 1901, but it is interesting to note that the deaths from gastric ulcer since 1901 were twice as many in females as in males. The death-rates are not necessarily a measure of the prevalence of the affection in the two sexes, but they are a good deal better measure of it than figures based on the diagnosis of gastric ulcer during life, and the mortality of two women to one man confirms what, I think, is the opinion of most clinicians that gastric ulcer is undoubtedly most common in the female sex.

*Cirrhosis of the Liver*

caused 8 of each 1000 deaths in 1905. In the

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five years 1866–1870 it caused 41·9 deaths per million living, and the deaths have gradually increased since that time to 134 per million in 1896–1900, though in the five more recent years, 1901–1905, it is satisfactory to find it decreasing to a rate of 121·2 per million.

A reported statement that the post-mortem appearances of the liver of the engine-driver killed in the Shrewsbury railway accident in 1907 showed that he had not been a teetotaler, but had not taken excess of alcohol, led to Dr. Donkin raising the question in the 'British Medical Journal' of November 21st, 1907, whether alcoholism produces cirrhosis of the liver? Subsequent writers in the journal questioned this strongly: one adduced as evidence that he had not found cirrhosis of liver in a large succession of inebriates; another gave instances of cirrhosis in undoubted teetotalers, and in other animals than man; and another writer urges that cirrhosis is due rather to syphilis than alcohol.

No one, so far as I know, has denied that cirrhosis may arise from other causes than alcohol; as long ago as 1871, Walshe ('Diseases of Lungs,' p. 535) wrote that "the liver of the cow exhibits as perfect specimens of cirrhosis as seen in the human subject"; nor has anyone insisted that alcoholism necessarily gives rise to symptoms of cirrhosis; the syphilitic form of cirrhosis is generally recognised.

None of these arguments disprove the belief that alcohol causes cirrhosis; as well might it be urged that because nephritis may arise from other causes than scarlet fever, or because it does not occur in all cases of scarlet fever, or because it occurs in other animals than man, or because a form of it may arise from syphilis, that scarlet fever does not cause nephritis.

I append a table showing side by side the mortality from cirrhosis and from alcoholism from 1866 to 1905, in quinquennial periods.

| | Deaths from cirrhosis per million living. | Deaths from alcoholism per million living. |
|---|---|---|
| 1866–1870 | 41·9 | 35·4 |
| 1871–1875 | 71·9 | 37·6 |
| 1876–1880 | 109·6 | 42·2 |
| 1881–1885 | 119·6 | 48·2 |
| 1886–1890 | 122·2 | 56·0 |
| 1891–1895 | 120·0 | 67·8 |
| 1896–1900 | 134·6 | 85·8 |
| 1901–1905 | 121·2 | 78·2 |

This table shows that the deaths from cirrhosis of the liver increase *pari passu* with those from alcoholism, and in the only instance in which the alcoholic deaths decreased, so also did those from cirrhosis of the liver. Taking this with the fact that the deaths from cirrhosis are about six times as frequent in persons engaged in the liquor trade as in the rest of the population, I think we may rest assured in the belief begotten of clinical and post-mortem experience that intemperance in alcohol, though not the only cause, is far the most potent cause of cirrhosis of the liver.

*Diseases of the Nervous System*

produced 80 of every 1000 deaths in 1905 as compared with 150 in a thousand in 1855, but it must be remembered that until the year 1900 all cases of apoplexy, cerebral embolism, and hæmorrhage were classified with diseases of nervous system; since 1900 they have been transferred to the group of diseases of blood-vessels. Diseases of the nervous system include many that we for the present call functional, and their prevalence is not shown or measured by death statistics.

A word, however, may be said as to convulsions, insanity, and tubercular nervous disease.

*Convulsions and epilepsy* together caused 30 out of the 80 per thousand deaths from nerve disorders; some 99 per cent. of these cases were in children under five years of age. Deaths certified as from convulsions are lessening year by year, as more care is taken in ascertaining the cause of the convulsions and certifying the death in accordance.

Deaths from epilepsy decreased steadily from 115 per million living in 1871–1875 to 86 per million in 1890–1900, but have since risen in 1901–1905 to 88 per million, probably because some deaths from what at one time would have come under convulsions have been put down as epilepsy. The Local Government Board returns show a great increase of lunacy, but there is no increased mortality from insanity shown in the death returns, probably because deaths of insane persons often occur from intercurrent disease. Deaths from general paralysis of insane have been stationary for the last four or five years.

*Tubercular meningitis* in common with tuberculosis of other organs has much lessened in fatality.

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*Deaths Annually per Million Living from Tubercular Meningitis.*

| Decennium. | Deaths. |
|---|---|
| 1861–1870 | 301 |
| 1871–1880 | 284 |
| 1881–1890 | 234 |
| 1891–1900 | 216 |
| 1901–1905 (5 years) | 185 |

The reduction was 40 per cent. in 55 years, much the same as in tubercular abdominal disease, but less than in pulmonary tuberculosis.

*Diseases of the Urinary System*

accounted for 30 of each thousand deaths in 1905. Of these 30, nephritis caused 24, while the other 6 arose from affections of bladder and prostate, calculus, etc.

*Annual Death-rate from Nephritis per Million Living.*

| Decennium. | Deaths. |
|---|---|
| 1861–1870 | 113 |
| 1871–1880 | 201 |
| 1881–1890 | 286 |
| 1891–1900 | 327 |
| 1901–1905 (5 years) | 379 |

There has thus been a steady *increase* in the fatality from nephritis, amounting to 220 per cent. in 55 years, a greater increase than in any other affection, with the exception of diabetes. Since the year 1875 deaths from acute have been separated in the tables from those from chronic nephritis, and show that the increased mortality is shared equally between the two.

The mortality is far highest among males, and of late there has been an increase in the deaths from nephritis among children under five years of age. Some of the increase is possibly due to improved diagnosis, but no great proportion can be set down to this cause, for nephritis in the *stage to cause death*, with which alone these figures deal, was little likely to be overlooked, of late years at any rate. Part of the increase is doubtless due to some cases of cerebral hæmorrhage being now certified under the head of chronic nephritis. But allowing for all this there is an undeniable important increase in the mortality, and presumably in the prevalence of nephritis, both acute and chronic.

The increased number of deaths in young children is noteworthy, though scarlet fever, believed to be one of its chief causes, has greatly diminished.

The great bulk of deaths from nephritis occur after the age of 55 years: after 75 years of age the deaths per million persons living at that age are nearly as numerous as the deaths per million at all ages together under 55 years.

It seems, indeed, that nephritis is one of the affections which has increased, and is likely to increase in fatality, with the greater longevity resulting from improved social and sanitary conditions.

*Calculus* and other diseases of the urinary system, bladder, prostate, etc., are unimportant as causes of death in comparison with nephritis, accounting for only 6 of 1000 deaths in 1905: these diseases appear to be gradually lessening in severity of late years, though allowance must be made for the fact that tubercle of genito-urinary organs has been transferred to the head of "tuberculosis" in the Registrar's returns. But the main important fact in genito-urinary diseases is the increased fatality from nephritis.

(P.S.—In 1906 there was a further increase to 387 per million living.)

*May 4th*, 1908.

(*To be concluded.*)

## A CLINICAL LECTURE

ON

# A CASE OF ABDOMINAL SUPPURATION OF OBSCURE ORIGIN, ON THE LEFT SIDE, WHICH WAS ULTIMATELY FOUND TO BE DUE TO DISEASE OF THE APPENDIX.

Delivered at the Bristol General Hospital.

By CHARLES A. MORTON, F.R.C.S.,
Professor of Surgery in University College, Bristol; Senior Surgeon to the General Hospital and the Children's Hospital; and Consulting Surgeon to the Cossham Memorial Hospital.

GENTLEMEN,—A boy, æt. 12 years, was admitted to the hospital on July 16th with the history of seventeen days' severe illness, which began with pain in the *left* abdomen and vomiting. Both symptoms continued, but the vomiting was not frequent. There was at times diarrhœa, at others constipation, and at other times again only mucus was passed. There had been no previous attack. On admission, in the evening, his temperature was 102° F., pulse 120, respiration 30, and when I

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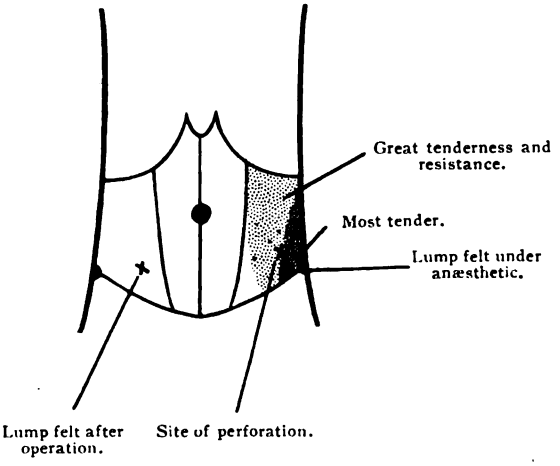
examined him next day I found the whole abdomen much distended, but especially the left side, and there was hardly any abdominal movement with respiration. It was resonant everywhere, but rather less so on the right side. There was no definite lump, but there was resistance and great tenderness in the shaded area in Diagram 1, and the greater tenderness was where the shading is deepest. On rectal examination there was general tenderness on pressing upwards, but no swelling was felt. I operated that afternoon. When under the anæsthetic I could feel a lump in the place marked (Fig. 1). I opened the peritoneal cavity through the outer border of the left rectus, at the level of the umbilicus, and thick pus and a little fæces escaped from a cavity between the parietal

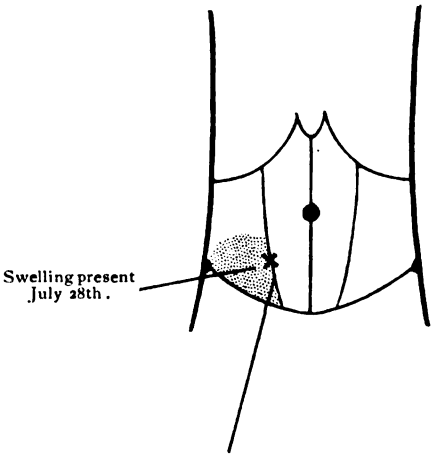
FIG. 1.




peritoneum, and some coils of intestine adherent to it, and into this space was a minute perforation from one of the adherent coils of bowel, from which light-coloured liquid fæces escaped. This perforation lay in the lower part of the abscess sac, near the point marked in the diagram. After the pus had been evacuated the lump felt under the anæsthesia had gone, but another small lump in McBurney's spot could be made out. I packed the abscess sac with iodoform gauze so as to stop the fæcal leaking. I adopted the left semilunar line for the incision as perhaps the best to explore the whole tender and rigid area, and was fortunate in cutting right into the abscess. So far it seemed like an abscess from perforation of the gut, but there was no evidence as to what that perforation was due to. There was no sign of tubercle in the peritoneum. I thought, however, the lump on the right side might be some indication of tuberculous deposit in the abdomen.

The temperature fell to normal just after the operation, but was up to 101° F. again next evening (see chart), and continued to rise higher every evening for some days. The gauze packing was removed four days after the operation, and there was never any further fæcal leaking. A week after the operation he had very alarming symptoms. He began to expectorate offensive thick pus, and developed very serious dyspnœa, especially if turned towards the right side, and we found an area of dulness at the left base, from behind which the house-surgeon aspirated some ounces of stinking pus. This relieved the urgent chest symptoms,

FIG. 2.




and the temperature began to fall, but it soon rose again, and as the expectoration of pus had ceased but the dull area persisted, on the 26th we ventured to give him chloroform and the house-surgeon opened the chest in the dull area, but did not find any pus, though he explored the interior of the lung as well as the pleural cavity. There seemed to be some adhesion at the base of the lung. A drainage tube was inserted, and two days later stinking brown pus began to discharge, and his temperature fell to normal (see chart). No swelling below the left costal margin could be detected at any time. By July 28th the abdominal abscess cavity had become obliterated, and the wound was only superficial. There had never been any further fæcal leaking after it had been opened and packed with gauze. But at this date I found a new swelling in the

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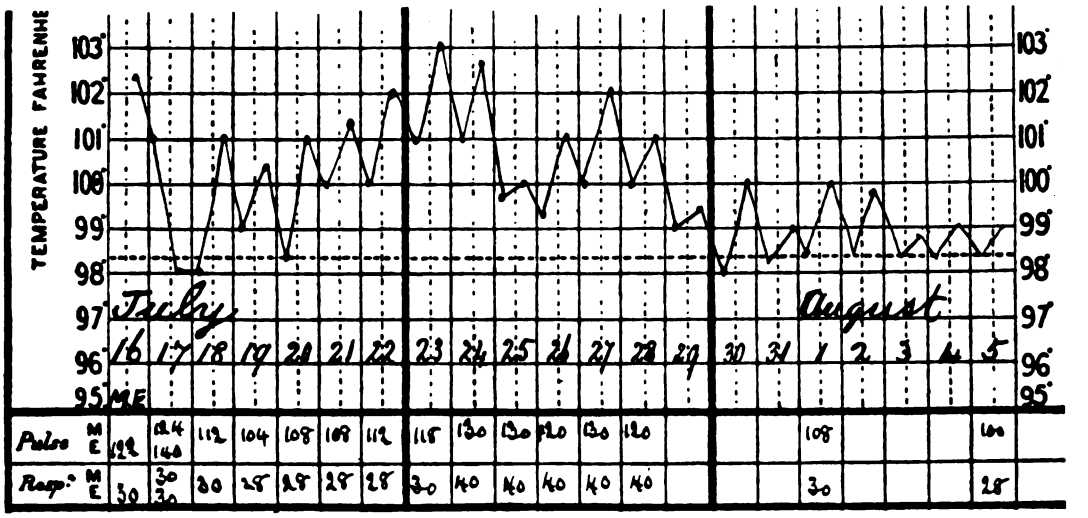
abdomen, as shown in Fig. 2, and this swelling was firm and tender and could also be felt *per rectum*, where it was tender also. I stated in my notes made at that time that the swelling "might very well be an appendix abscess." I should have operated on it if his chest condition had not been so bad, but the large swelling gradually subsided, leaving, however, a little ill defined lump at the point indicated in the diagram, and a general thickening and lumpiness of the pelvic roof as felt *per rectum*. On September 3rd he was discharged. There was only a small superficial sinus in the chest wall, but there was some persistent dulness and deficient air entry, quite at the base in the region of the sinus. The abdominal wound was practically healed. The little lump (where shown in the diagram) was even less marked than before.

I did not see this boy again until he was re-




admitted on October 7th, with a history that he had remained quite well until 8 A.M. that day, when, during defæcation, pain came on in right lower abdomen, and he vomited several times that morning, and in the afternoon a doctor who was called in found his temperature 102° F. He was admitted in the evening. He had frequent, but not painful, micturition all day. A swelling was found almost in the centre of his abdomen. The bladder was found to be empty when a catheter was passed. Next day I found the swelling as shown in Fig. 3. In shape and position it was very like a distended bladder, but was not quite as dull, and there was resonance between it and the pubes. On the right side of the central mass, and placed much more deeply, was a firmer and more tender swelling (shown in Fig. 3). Nothing of the swelling could be felt *per rectum*, but the pelvic roof seemed rather thickened. The condition of the base of the lung was now practically normal. It seemed to me there must be some fresh suppuration, and the same afternoon I made an incision through the outer border of the right rectus, below the umbilicus, and on dividing the peritoneum evacuated a little clear serous fluid, and came on a mass of adherent coils of bowel wrapped up in the omentum, and this formed the central swelling. The true pelvis was shut off from the mass by adhesions. I could recognise part of the cæcum protruding from the right side of it. I packed off the free peritoneal cavity around it, and broke down the adhesions which bound the omentum and coils of bowel together, and found in the deep parts fairly recent lymph, and deeper still but on the left side an ounce of stinking pus, about the level of the top of the sacrum. I then searched for and found the root of the appendix coming off from the cæcum, and could trace it to the pelvic brim, along which it seemed to lie, with the tip towards the left, but the tip did not seem to me to actually reach the left side of the abdomen. I had to tear it out of old adhesions in order to remove it. It was obliterated half an inch beyond the junction with the cæcum, and there were two perforations beyond the seat of obliteration. The suppurating area was drained, and the swelling formed of the adherent coils steadily subsided, and a month later there was only a small granulating area in the abdominal wall, and he was discharged soon after.

Looking back at the condition present in the first illness, in the light of what we found in the second, I am inclined to think that all his troubles were due to disease of the appendix. The abscess on the left side, opened at the first operation, may have been one of those abscesses we sometimes find away from the appendix, when the peritonitis has been somewhat diffuse, and pus is left shut up by adhesions beyond the area of the appendix, and not directly communicating with it: and there may

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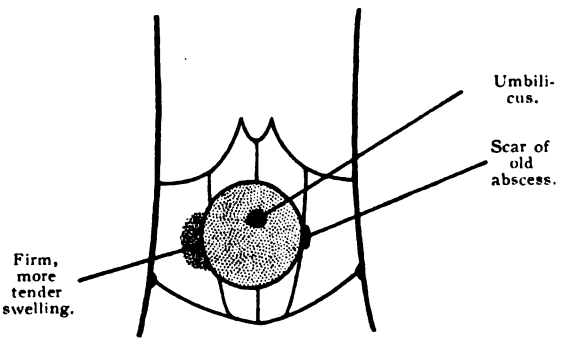
have been another, deeper abscess tracking up to the diaphragm on that side, formed in the same way, from the spread of a suppurative peritonitis up the left side of the abdomen, though starting in the region of the appendix, just as finally we found the chief mischief placed centrally, and not extending up the right side, because the appendix lay on the pelvic brim on the inside of the cæcum. Such an abscess may have given rise to the physical signs at the left base, and by eroding the diaphragm, have penetrated the base of the left lung, and caused the expectoration of the offensive pus which it contained. Moreover, the inability to find pus in the left pleural cavity when opened, although pus had been aspirated off two days before, was evidence in favour of the view that the pus was sub-diaphragmatic, and its brown stinking character was strongly suggestive of its abdominal origin. No doubt it eventually burst through the adhesions at the base of the lung found when the pleura was opened. The aspirating needle may have passed through the dome of the diaphragm into a sub-

FIG. 3.




phrenic abscess. But an abscess just below the diaphragm may be associated with an empyema just above, and this combination may have existed in this case, the empyema being limited to the space between the base of the lung and the diaphragm. We shall never know for certain exactly where the pus was. I know, from my experience of another case, that you may have a large abscess in the abdomen, lying posteriorly in the splenic region, without any swelling under the left costal margin, and no other definite signs of its presence, and that such an abscess may eventually burst through the diaphragm into the bronchial tubes, and cause the death of the patient from the pus rapidly filling the bronchial tubes of both lungs.

It is, of course, well known how often disease of the appendix will cause a pelvic abscess, but it is certainly rare for it to cause abscess on the left side. I have operated on one other case myself. A boy had an abscess on the left side, near the sacro-vertebral junction, and the appendix lay across the top of the sacrum, with the tip perforated on the left side. A pelvic abscess from appendix disease may also track up the left side.* There may also be a transposition of viscera, so that the cæcum and appendix may be on the left side. In this case the heart would be on the right side.

When I opened the first abscess and found a perforation of the intestine I regarded it as the cause of the abscess, as one would naturally do, but now I am inclined to think it may have been only a secondary condition due to erosion of the pus from without.

Mr. Battle, in his book on disease of the appendix, records a very remarkable example of left empyema, with a sinus which persisted for twenty years, and at a post-mortem examination disease of the appendix was found to be the cause, a sinus being present from it to the pleura.

The fact that in my case the pain started on the left side is interesting. One would have expected a right-sided pain, or a pain across the lower abdomen, or a central or epigastric pain at the commencement, but not a left-sided one, even though an abscess was eventually formed on that side. Since operating on this case I have had another, in which the pain began on the left side. The patient was a particularly intelligent boy, and was quite clear that the pain seized him in the left lower abdomen. He was seen by the doctor a few hours later, and then the tenderness was over the region of the appendix, and when I saw him in the evening the pain and tenderness were there also, but curiously enough the cutaneous hyperæsthesia was over the left lower abdomen. The case was one of diffuse peritonitis over the lower abdomen and pelvis, starting from a diseased appendix, which lay on the inside of the cæcum.

In the case which I have made the subject of this lecture no doubt the persistent though very slight swelling in the appendix region between the attacks was due to persistent mischief in the appendix, and looking back it is easy enough now to see that it would have been wise to have operated on it. But it is so easy to judge rightly with new facts before you what you should have done when your evidence was imperfect, and I hope this case may be as instructive to you as it has been to me, in teaching how suppuration of obscure origin in the left side of the abdomen may be connected with a diseased appendix, thought not at first obviously so.

(The temperature chart gives the temperature to the end of the third week. There were occasional rises to 99° and 100° F. after that date, and once to 101° F.)

*May 4th*, 1908.

* Since this lecture was delivered Mr. Barnard has recorded a case of expectoration of foul pus, associated with sub-diaphragmatic abscess on the left side, which was an extension of a pelvic abscess, due to appendicitis. Mr. Barnard points out how such a pelvic abscess may track up, either inside or outside the ascending colon. In his case the sub-diaphragmatic abscess caused a marked abdominal swelling and was opened through the abdominal wall.

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# THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE, AND CLINICAL REVIEW.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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## A CLINICAL LECTURE

ON

# GENERAL PARALYSIS OF THE INSANE.

Delivered at St. Bartholomew's Hospital.

By W. P. HERRINGHAM, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—When you study disease in books, as you must do a great deal for examination purposes, I think you ought not to forget that Nature has not set the question in the same way. You get a wrong impression of how the disease will come before you when it comes embodied in a patient. For instance, you go up to an examination hall, and the examiner sets you down to the question, "Give a description of the onset and course of general paralysis of the insane"? You will probably be able, very plainly and very fully, to tell him how general paralysis of the insane begins, sometimes with symptoms of exaltation, sometimes with symptoms of melancholia, sometimes with symptoms of dementia, and sometimes with paralysis. But Nature never asks you, how does general paralysis of the insane begin? or, what is the cause of general paralysis? She never sets the question like that in the least. I am tempted to recall to you old times and remind you how, in the Odyssey, Athene used to present herself to Ulysses, sometimes like a young man, sometimes like an old woman, sometimes like one of his own friends. Nature plays just the same tricks. You remember how Ulysses explains to Telemachus that this sort of thing is quite easy for the gods who dwell in the high heavens. Nature comes before you sometimes as a member of the City police, sometimes in the guise of an anxious wife, perhaps a little the worse for drink, and sometimes she sends a note by Hermes, who brings it from a neighbour-

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ing doctor. And what she says is, "I picked up this man on Blackfriars bridge in a fit, sir." Or, "Please, sir, my husband came home with a bad headache last night and he was sick after his supper." Or, "My dear sir, I was summoned to this patient and found him unconscious." And then she says simply, "What is the matter with this man?" She does not say "What is the course of general paralysis?" And if you have been learning your work very diligently in books and got ready an answer to the examiner, you have to unship the whole of the labels which you have been hanging up round your mind, and throw them into different heaps before you can answer any of the questions which are put to you in real life.

On January 31st, a man, A. P—, æt. 31 years, a porter in a warehouse, was brought here with the statement that he had had a fit in the last few days. And the doctor wrote a note with him, saying he had seen him in similar conditions before and he had usually found him unconscious and paralysed on the right side. When a man tells you that he usually finds a patient paralysed on the right side and unconscious, it is rather a striking thing. In the first place, it is evident that the paralysis passes off quickly, because the man was not paralysed when he came to us. And the same thing must have happened several times before. So the problem appeared very difficult, and after a great deal of inquiry we came to this: that on Easter Monday, 1907, he had an attack of loss of memory, which had lasted five hours consecutively; that two days later he had had a fit; that next day he went to work, so the fit could not have been anything which incapacitated him for long. In the evening he had another fit; but he went to work again the day after. He had had several attacks of numbness in his arms, which had temporarily prevented him from working. He never had another definite fit until three weeks before coming to us, and then he had a third fit, typically epileptic in character; that is to say, he had fallen with a cry and had been completely unconscious, he had had convulsions, and he passed a motion under him. We noticed when he came in that his speech was not quite like that of an ordinary man. He talked a little thickly and sometimes stumbled over one or two letters. And when we asked his wife whether that was his natural speech she said, "No, it was not," and that "she noticed an alteration in it since the first fit last Easter time." We asked her also if she had noticed any change in his mental condition, and she said, "Yes, he had been, since that first fit, a little more lethargic and apathetic than he naturally was." And then when we came to examine him we found that, besides this little thickness in his speech, his tongue was somewhat tremulous. We got a good jaw reflex, from tapping the lower jaw with the mouth open. But that, of course, is not very remarkable. We found that his pupils were equal, and though they contracted to light they dilated again; that is to say, they did not remain contracted. There was no ocular palsy, and the optic discs were natural. There was no weakness in any of his limbs, and his deep reflexes were natural. There was no alteration in sensation anywhere, nor any alteration of the superficial reflexes.

When a case of that sort is brought with such very slight symptoms I suppose the first thing which occurs to everybody is this: is the case organic at all? Is it functional? And one would have been tempted to say it was perhaps functional, except that he had had a most definite epileptic fit. A definite epileptic fit is incompatible with hysteria; an attack in which the patient entirely loses consciousness, and passes his motions under him is incompatible with functional disease. And the next point which was considered was, was it a case of epilepsy? Epilepsy does occasionally leave temporary paralysis, but, so far as I know, it never leaves a certain thickness in speech, nor, except in its later stages, does it alter the mind. So we could not think that those symptoms, slight though they were, could be accounted for by epilepsy, nor does it ever produce that alteration of pupils which I remarked upon. We were therefore forced to confess that, slight though the symptoms were, they must be organic. And then the question arose, what sort of organic disease was it? It was not acute inflammation, nor any vascular disease. Because how could a man who had had thrombosis, or cerebral hæmorrhage, or cerebral embolism, recover three or four times within a few hours? It was impossible with a vascular lesion, so you must rule that out. Nor could you think it was cerebral tumour. He had none of the cardinal symptoms of cerebral tumour, such as vomiting, or optic neuritis, or headache, he had not even any of the bye-products of cerebral tumour, such as local

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paralysis. There was nothing which would lead you to diagnose a cerebral tumour. So we were driven, if we believed it to be organic disease, which we did, to think that it must be some form of degeneration. And the first thing which occurred to me was the degeneration called general paralysis of the insane, which, as you know, is very much the same sort of thing as the sclerosis of the kidney which occurs in chronic interstitial nephritis, in which, what pathologists call the nobler tissue, perishes and becomes atrophied, and lower tissues, partly to take their place, and partly as a result of the irritation caused by the dead tissues, are increased. But before we could say it was general paralysis of the insane, we had to think of something else. Because there is another disease, disseminated sclerosis, which produces symptoms not at all unlike those of general paralysis, as I shall show you in this next case. This patient,* W. B—, is a man, æt. 27 years. He does not give a very good history. He is rather dull, and whether that condition is one which he has had all his life I cannot quite say. But the history which we got from him and from his friends is that three years ago he was a labourer on a railway and had a bad fall. Since then his right arm has been weaker than his left. Eleven months ago he had a fit, in which he was unconscious for five hours, and since then his legs have been weak. When he walks, as you see, he draws his feet along the ground. He has a spastic gait, and he has a defect in his speech. His impediment of speech is very difficult to distinguish from that of the other man. He is much steadier than he was when he first came in, and he touches a given point with his finger very fairly. We also found that he had on the outer side of his right ankle an anæsthetic area. He had, in addition, slight spasmodic torticollis, but that has been much better since he had a bad tooth treated. He has the rotatory form of nystagmus, which is very unusual. Besides that, his knee-jerks are brisker than natural, and he has slight ankle-clonus. There is also contraction of the fields of vision of both eyes, and some optic atrophy. The two cases, that is to say, his case and the man I first spoke of, present many points of resemblance. Both the cases began with fits, and perhaps W. B—'s fall may itself have been due to a fit, both have some mental change, and a certain amount of paralysis, for A. P— had some tremor of the tongue, and had had hemiplegia. Both of them have an alteration in speech. The symptoms of W. B— are much worse than those of A. P—, and yet I feel convinced, as did everyone else who saw him at the consultation, that this man you have just seen has disseminated sclerosis, and A. P— has general paralysis of the insane. How can you form a diagnosis in cases such as that? This last man, W. B—, has symptoms which would be quite consistent with general paralysis of the insane. For instance, he has dulness, fits, and paralysis, and defect of speech. But, in addition, he has spasticity of gait and optic atrophy and nystagmus. The first of these is uncommon in general paralysis, the second is very rare, except in the very last stages, and nystagmus practically never occurs in it. So you have three symptoms which, cumulatively are impossible to reconcile with the diagnosis of general paralysis of the insane. You must allow that he has something else, and since the only other diagnosis possible is disseminated sclerosis, that is what the diagnosis was. The first man had symptoms which were consistent with general paralysis, but he had nothing which was not consistent with it, and there was no call for a diagnosis of anything else. That diagnosis satisfied his condition, and there was nothing which was inconsistent with it. So the only difficulty about it was that the case was very early and his symptoms very slight. I remember in the wards on various occasions a few other very early cases which were not quite so difficult as this, but almost. We had a man called William M—, æt. 47 years; he had been depressed for several years and said he felt quite incompetent for his work, he could not "buck himself up" or force himself to do his business. He believed he would end in the workhouse. He had had attacks of temporary weakness of the right leg and of aphasia, and his pupils were uneven and did not react to light. He had had syphilis twenty years ago. Another man was Charles T—, æt. 40 years. Four years before he had had a fit. Nine months before he had influenza, and was "off his head" for six weeks. Since then his memory had been very uncertain, he had not been able to work, and "he had turned rather religious."

* The patient was shown.

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Afterwards the right leg suddenly became weak and he fell, but it was all right again subsequently. He talked a little thickly, but there were no delusions and no tremor. His pupils were unequal. He denied having had syphilis, but perhaps in his unregenerate days he would not have denied it. Another case was Edward E—, æt. 34 years, who started in business for himself; he was a grocer, and had great ideas of making a fortune, but his business proved to be a bad investment, and he had been depressed for a time. He got over the depression and later was in his natural temper, but a month after complained of pain in his back and legs, and three weeks before that time his walking became unsteady. The pupils were unequal and did not react to light. Then we had a ship's captain, æt. about 40 years, and I remember he was admitted in consequence of extreme ataxia. Some of you may have seen those cases of acute locomotor ataxia. I have had two in the last three years, in which the symptoms come on in two or three months, and patients are extremely ataxic when they come. This man's ataxia had come on during his last voyage to London. We thought at first that he had nothing but this ataxia. But after he had been in three weeks he developed most wonderful delusions; they were very marked indeed, and he was obviously a case of general paralysis of the insane beginning with ataxic symptoms. We had another case. A man in responsible employment, æt. 48 years, was one day missed from his post, and was brought back by the police, who found him wandering about London unable to give an account of himself. He was rather emotional when brought in, his pupils were unequal, and his grasp extremely weak. One could not believe that his weakness was due to deficiency in the muscles, because he was a well-developed, muscular man; but his squeeze was that of a child. His knee-jerks were very brisk, and his wife stated to us that he had not been like himself for about three months. He got slowly better, but he never regained his usual condition of mind. He was, later, still a little emotional and looked very sad. He is now able to do ordinary, but not very responsible, work. How long that condition will remain we do not know. You see those were cases in which there was obvious mental change which could not be got over, and there was evidence of organic disease of some sort—paralysis, ataxia, or alteration of pupils. The only difficulty about that consisted in this, that the symptoms were so slight that you might have felt inclined to say they might be accidental. That is the reason why I have brought the case of this man, A. P—, before you to-day: in order to impress upon you that accidental is a term you ought never to use. His symptoms were extremely slight—a very moderate blurring of the speech, not nearly so much as in the second man, a slight alteration in the mental condition, and a history of fits, of which no trace remained. And yet the only possible diagnosis, when you thought it out, was that he had the beginnings of the most serious disease that a man can have. And that is really the lesson of this lecture—never slur your diagnosis. I believe, honestly, that the difference between the good doctor and the bad doctor is that the good doctor is never content until he can satisfy himself that his diagnosis will account for every symptom in the case, or else that there are some symptoms which he cannot explain; in that case he states to himself exactly what they are, and that he must wait for further evidence until he can give an explanation. He books them as at present unexplained, and does not do what we are all so much tempted to do, such as saying, "Oh yes, he has had some fits, and no doubt that has left a little damage to the cortical tissue." That is the sort of diagnosis which you might be tempted to give to the patient's friends, and it might satisfy them, but it ought not to satisfy you. Such a condition, when you think it out, is seen to be impossible. Fits must be due to something, and either they do show evidence of organic disease, or they do not. They do not leave the cortical tissues damaged unless there is some definite reason for it. I should say that the difference between a good doctor and a bad doctor is this: that the good doctor makes up his mental accounts accurately, whereas the bad doctor is content to have a large item which he calls "sundries."

*May 11th*, 1908.

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We have received from Messrs. Cassell & Co. a copy of Dr. H. Campbell Thomson's book on 'Diseases of the Nervous System.' The volume is embellished with 8 coloured and 12 black-and-white plates, and 101 figures in the text.

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## A CLINICAL LECTURE

ON

# SYPHILITIC DISEASE OF THE NERVOUS SYSTEM.

Delivered at the West End Hospital for Nervous Diseases.

By HARRY CAMPBELL, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—This patient is æt. 36 years. He gives a definite history of syphilis. I shall put before you the salient features of his case, which, I may remark, were not elicited until I had spent a couple of hours in going into it. I need hardly remind you that considerable discrimination is needed in getting at the essential points of a case. Not only must the physician know what to look for, and what to ask for, but he has to separate the grain from the chaff. Once the main facts are obtained, the diagnosis is often comparatively easy; otherwise it may be very difficult. It is for this reason not easy to test the clinical acumen of a candidate at examination by getting him to write a commentary on a recorded case in which all the essential features are set forth.

The patient in February of 1906 had an attack of what he calls "blankness," lasting twenty-four hours, *i. e.* he sat or lay all this time looking about him without being able to recognise anybody, even his own wife. When he came round he had no recollection whatever of the attack. Some hours after he regained consciousness he was seized with severe headache. This lasted on and off for a fortnight. He then noticed weakness in his left leg. About four weeks after the initial seizure he went into hospital for a month, and during this time the weakness in the left leg increased to complete paralysis, while the left upper extremity gradually became paralysed, and some weakness in the left side of the face appeared. He tells us that during his stay in hospital he took iodide of potassium: he is a man of inquiring mind, and he is quite sure of this. On leaving the hospital he had the iodide mixture made up and continued to take it until August, 1906. His hemiplegia had greatly improved by this time, and he was able to get about, but he did not consider himself fit for work until April, 1907, when he went back to his occupation as clerk. After he had been at work for about a month he was seized with violent pains in the lower region of the back and a feeling of constriction round the waist, and about the same time he noticed a fluttering sensation in the right heel and a tingling, burning sensation in the right thigh. Such, in brief, is the history of the case.

Now for the examination of the patient. The left upper extremity has almost completely recovered power. As you see, he can grasp my fingers firmly, and he can bend his hand and fingers well back. This plan of getting the patient to extend the wrist and fingers is a good test of the degree to which the upper extremity has recovered in hemiplegia. If the patient can completely extend wrist and fingers at one and the same time, the strong probability is that recovery is practically complete. When the hand is allowed to fall down at right angles with the forearm it may be comparatively easy, even with considerable paralysis, for the patient to extend the distal phalanges, because in this position a moderate contraction of the long extensor suffices to extend them; but if the patient can extend the fingers in the extended position of the wrist, it shows that he has considerable power over the muscle which is generally the most paralysed, and the last to recover, in hemiplegia. In very bad cases the patient may not be able, even after many years, to move in ever so slight a degree the distal phalanges, not even in the flexed position of the wrist.

You will observe that the "deep reflexes" are greatly increased, and that a brisk contraction is elicited by tapping almost any part of the upper or the lower extremity. Note also that ankle clonus and Babinski's sign are present. With this increase in the deep reflexes there is, as often happens in hemiplegia, an absence of the abdominal and cremasteric reflexes on the affected side.

Here, then, is a patient whose illness began with an attack of mental obscuration lasting several hours, and was followed by protracted headache, and after a few weeks by a progressive hemiplegia, beginning in the lower extremity and subsequently affecting the upper extremity and the face, and yielding to anti-syphilitic treatment. It is obvious that we have to do with a syphilitic lesion, and the question we have to ask ourselves is: What is

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the exact nature of the lesion? Much the most common cause of hemiplegia, occurring in a man under forty, is thrombosis of the middle cerebral artery from narrowing of its lumen by a localised deposit of gummatous material in its inner coat—"syphilitic thrombosis," as we may call it. In cases of this kind, however, the hemiplegia usually comes on suddenly: it is true that there may be premonitory symptoms, such as numbness and even temporary paresis, in one or other limb of the affected side, but a steadily progressive hemiplegia such as occurred in this man can only, so far as I know, be produced by one lesion, *i. e.* a new growth. Therefore I judge the lesion to be a gumma. Such a lesion by causing an increase in intra-cranial pressure might well have produced the initial attack of "blankness." The headaches from which the patient suffered are consistent both with the diagnosis of thrombosis and gumma: in at least half the cases of hemiplegia from syphilitic thrombosis the stroke is preceded, for several days or even weeks, by headache—a fact, I need hardly say, of great importance in view of prophylactic treatment. I have seen many cases of syphilitic hemiplegia in which this valuable clue has been neglected. Whenever a patient who has been infected with syphilis begins to suffer from unaccustomed headache, recourse should at once be had to vigorous anti-syphilitic treatment.

The diagnosis of gumma, then, somewhere in the cerebral motor tract (probably immediately under the cortex) explains the leading features of the case: the initial attack of blankness, the severe headache, the progressive hemiplegia, and finally the improvement which took place under iodide. Such weakness as is left on the hemiplegic side is probably due to permanent destruction, with secondary descending sclerosis, of certain of the cerebral motor fibres—a condition of things which cannot be ameliorated by iodide or mercury.

The symptoms which made their appearance in April, 1907, I judge to have been due to a syphilitic meningitis in the lower part of the cord involving the right side chiefly. Such a lesion would account for the pain in the back, for the feeling of constriction round the waist, and for the paræsthesia and weakness in the right lower extremity.

Here I would call your attention to a very interesting fact. From the time the patient first went into hospital up to the present date he has been taking iodide of potassium. It would appear that the cerebral gumma yielded to the iodide, and that while he was still taking this drug a fresh syphilitic lesion developed in the cord. "But," you may ask, "is it possible for a syphilitic lesion to start *de novo* while the patient is undergoing a course of iodide?" There is no doubt that this may happen. Let me in this connection cite a classical case recorded by Sir William Gowers: "Many years ago a patient came under my care with symptoms of a cerebral syphiloma. Under 10 grains of potassium iodide three times a day the symptoms rapidly passed away and the patient seemed well. He continued, however, to attend the hospital and to take the medicine. At the end of three or four months he presented symptoms of disease of the upper dorsal region of the spinal cord. They progressed rapidly and caused his death. It seemed impossible that the disease could be syphilitic, since he was still taking the medicine which had so speedily relieved all indications of such disease in the brain. But the necropsy shook our confidence in the seemingly impossible. In the brain there was the shrunken relic of the gumma; in the spinal cord invading it from the membranes on one side was a similar growth in the active stage, typical under the microscope and typical also in the naked-eye feature of disseminated caseation—a character of conclusive significance. The lesson this case taught me I have since seen confirmed by other cases—not my own, however. It is that treatment may lose its power if continuously maintained."

I shall recur to this question of treatment presently. Meanwhile let me recount to you the most common syphilitic diseases of the nervous system. The most common intra-cranial syphilitic lesions I see here are: hemiplegia from thrombosis, general paralysis of the insane (which is essentially an intra-cranial disease), and basal meningitis, manifesting itself by affection of the cranial nerves, notably of those passing into the orbit. A gumma forming a genuine cerebral tumour is, in my experience, distinctly less common than the three conditions just mentioned.

The most common syphilitic affection of the spinal cord is tabes dorsalis. (This may be considered as essentially a disease of the cord, though the brain is likewise affected.) Syphilitic meningitis and syphilitic sclerosis are in my experience less

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common than tabes. Acute myelitis is said occasionally to result from syphilis. I ought to have mentioned, when speaking of general paralysis of the insane, that there is a form of brain affection which may conveniently be spoken of as syphilitic dementia. There is loss of memory, and other evidence of mental breakdown; the patient often exhibits eye symptoms, such as inequality of the pupils, the Argyll-Robertson phenomenon, paralysis of the ocular nerves, or affection of the nerve; sometimes convulsions may occur. It is not easy to give a concise clinical account of this distressing disease, though a little experience enables one to identify it without much difficulty.

Now to return to the subject of treatment. How far does syphilitic disease of the nervous system lend itself to treatment? It may be said at once that among the intra-cranial syphilitic diseases syphilitic insanity—whether of the kind just referred to, or the more common form of general paralysis of the insane—and hemiplegia due to syphilitic thrombosis are wholly untouched by anti-syphilitic remedies. On the other hand, intra-cranial gumma and syphilitic basal meningitis are often amenable to treatment. As regards syphilitic affection of the spinal cord, tabes dorsalis is absolutely untouched by anti-syphilitic treatment, whereas syphilitic spinal menigitis, or gumma involving the spinal meninges or the cord, often yield to anti-syphilitic remedies.

How are we to treat the amenable cases? We should, of course, endeavour as far as possible to prevent an outburst of syphilis, and if we have the slightest warning of such an event we should at once give large doses of iodide or mercury, or both. Sir William Gowers is not in favour of large doses, nor, in view of the case in which a gumma developed in the cord whilst the patient was taking iodide of potassium, does he recommend a long course of the drug or of mercury. He prescribes 10 grains of the iodide with small doses of mercury, for, say, two months. Now, while I do not believe in prolonged courses of this drug in the tertiary stage of syphilis, I am strongly of opinion that it should be given in large doses. When I have reason to suspect the existence of a gumma, or of gummatous inflammation, in any vital part of the nervous system, I prescribe 1 drachm of the iodide four times daily, and I am very doubtful whether a fresh syphilitic lesion could develop while the patient was undergoing such vigorous treatment. In any case, tertiary syphilis of the nervous system has in my practice yielded much more rapidly to large than to small doses of the iodide. Thus, I recall the case of a patient sent to me for violent syphilitic headache. He was a prize-fighter, but all the pluck had been taken out of him by the headache, which, he declared, had prevented him from having a wink of sleep for three months. This, I imagine, was not possible, though there can be no doubt that his sleep had been greatly disturbed during that time. He came into my room holding his head with both hands, writhing with the pain. His scalp was exquisitely tender, as not infrequently happens in syphilitic headache, and as he gave a history of syphilis I had no doubt as to the treatment to be pursued. I wrote a prescription for a drachm of iodide, to be taken four times daily, and told him to go to the nearest chemist and to take a dose at once, and another at bedtime. Meanwhile I communicated with his doctor, and ascertained from him that the patient had for some time past been taking 10 grains of the iodide three times daily. Well, the patient took a drachm of the drug, *statim*, as advised, and another before going to bed. The effect was unmistakable. That night he slept soundly. The next day the headache was much better and continued to improve, until by the end of a fortnight it had entirely disappeared. This is but one of several similar cases, and on this account I advise you, whenever syphilis threatens any vital part of the nervous system, to give 1 drachm of iodide of potassium four times daily. If the symptoms do not yield rapidly, say after a week, I diminish the dose, or stop the drug altogether; otherwise I continue the medicine in undiminished quantity for six to eight weeks. It is astonishing how well patients tolerate large doses of iodide. If they do not, it is no great matter. What is the most virulent iodism compared with blindness, life-long paralysis, or death?

I now show you another patient. He is æt. 19 years, and follows the occupation of gardener. Fifteen months ago he had a bicycle accident, falling on his shoulder. Nine months ago he noticed that his left hand was weak. He now shows decided wasting in the thenar, hypothenar, and interosseal muscles of this hand, the fingers and thumb of which he can neither abduct nor adduct. Owing to paralysis of the interossei

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the proximal phalanges of the fingers are hyper-extended and the distal phalanges flexed, producing the well-known *main en griffe*. You will observe, too, he has little power in the extensors of the wrist, and that the flexors are also somewhat weak.

The condition of the "deep reflexes" is interesting. You will notice that the left wrist-jerk cannot be elicited, but this is not surprising seeing that the extensors of the left wrist are greatly atrophied. Otherwise most of these reflexes are exaggerated in the extremities, upper and lower, of both sides. Thus not only are the knee-jerks exaggerated, but a rectus clonus can be elicited, as also ankle clonus. The lower extremities also show a tendency to rigidity—hypertonus. It will generally be found that the ease with which the "deep reflexes" can be obtained rises and falls with the tonicity of the muscles.

Another noteworthy feature of this case is the presence of "fibrillary twitchings." These, though not actually pathognomonic, are highly suggestive of slow destructive action in the cell body of the lower motor neurone. They are not only present in the paralysed limb but in all four extremities. I have observed them, among other muscles, in the deltoids, pectorals, and vasti. Not long since I saw an old gentleman who had for eighteen months been suffering from atrophy of the small muscles of one hand. On getting him to strip I noticed that almost all the muscles of his body were affected with fibrillary twitching, and from this I concluded that the disease was not confined to the motor neurones of the paralysed hand but that it affected the entire system of the lower motor neurones. He has since died from a generalised atrophic paralysis.

Now as to diagnosis. The atrophy of certain muscles and the presence of widespread fibrillary twitchings shows that the lower motor neurones are affected. On the other hand, the hypertonus and exaggerated deep reflexes indicates involvement of the upper motor neurones. The patient is, in fact, suffering from degeneration of the entire motor tract. If the upper neurones alone were affected we should simply get, besides paralysis, hypertonus and exaggeration of the deep reflexes; and if the lower neurones alone were affected we should get atrophy only, and this would also be the case if both the upper and lower motor neurones of a muscle were affected, for the atrophy caused by the latter lesion would mask the symptoms due to the former lesion. Suppose, however, all the upper motor neurones, and a large proportion of the lower motor neurones of a particular muscle, *e.g.* the biceps, to be affected, many of the fibres of this muscle would be in a state of atrophy, while the others would be hyper-tonic, and the atrophied, hypertonic muscle would give exaggerated deep reflexes, as happens with several of this patient's atrophied muscles.

This condition of things may frequently be observed in progressive muscular atrophy. It is now known that in most cases of this disease both the upper and lower motor neurones are involved. The case just shown you is an example of Charcot's "amyotrophic lateral sclerosis." In his classical descriptio n of this disease Charcot makes three stages—an initial stage characterised by atrophy and increased deep reflexes of the upper extremities, a mild stage in which there is added spastic paraplegia from involvement of the upper motor neurones of the lower extremities, and a final stage in which symptoms of bulbar paralysis appear.

Now what is the pathology of this disease? How are we to account for such a widespread degeneration of the motor system? Is it due to an intrinsic weakness of the affected neurones, one *i.e.* germinal in origin? Or is it caused by the action of some pernicious influence operating upon the neurones from without. This question we cannot at present answer positively, but it seems to me highly unlikely that myriads of neurones belonging to a special system should at one and the same time spontaneously take it into their head, so to speak, to misbehave themselves in exactly the same way. To me it seems much more likely that their degeneration is induced by some noxious influence operating through the plasma.

I regret to say that we have no control over this disease. It tends to get steadily worse and to end fatally two to three years after its commencement.

I next propose to direct your attention to a case of pseudo-hypertrophic paralysis. This disease appears to be due to some developmental defect in the muscular system in consequence of which the muscular fibres atrophy, this atrophy being accompanied by an abundant growth of fibrous and fatty tissue between them. Observe what large calves the boy has. They feel as firm and

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hard as cork. This is due to the fact that the sheaths of the great muscles of the calf are tightly packed with fibro-fatty tissue. You will notice that the child has talipes equinus from contraction of the calf muscles; also that the knee-jerk is absent. This is characteristic; only in exceptional cases is it preserved in this disease. Its absence is due to affection of the quadriceps. You will observe that portions of this muscle are atrophied, while others are in a condition of pseudo-atrophy, *i. e.* in some parts there is atrophy of the muscular fibres without any over-growth of fibro-fatty tissue, while in other parts the atrophy is accompanied by such over-growth. The buttocks, as not infrequently happens, are large, and you may notice that the erectores spinæ stand out with unusual prominence, although these muscles, as well as the glutei, are manifestly weak. There is complete absence of the sternal portion of the pectoralis major—quite a common feature in this disease.

I should like to draw your attention to the fact that in all the "amyotrophies" there is a tendency for the chest to flatten when the trunk muscles are affected. This results from paralysis of the inspiratory muscles whose contractions determine the mean size of the chest. Throughout life these muscles are continually on the watch so to regulate the mean thoracic capacity that the lungs shall be kept moderately on the stretch, and thus maintain a negative pressure round the heart. Now inasmuch as the inspiratory muscles in this boy are degenerated, the thorax is subnormal in size. As a result the lungs are not properly expanded. Hence there is both a narrowing of the pulmonary vascular bed and a diminution in the normal suction action of the lungs on the heart, and in consequence of this the circulation is being carried on at a disadvantage. I have watched many cases of pseudo-hypertrophic and other forms of amyopathy from this point of view, and have noticed that towards the final stage there is considerable interference with the circulation, as shown by the rapid action of the heart and sometimes even by the occurrence of dropsy. By the time this stage is reached the patient is bedridden, but even when he is propped up in bed the circulation is carried on with great difficulty. In the upright position it could not be maintained for any length of time.

*May 11th*, 1908.

# A REVIEW OF MORTALITY STATISTICS DURING THE LAST HALF CENTURY.*

By SIDNEY PHILLIPS, M.D., F.R.C.P.,
Physician to St. Mary's Hospital.

(*Concluded from page* 61.)

*Infectious diseases*

accounted for 62 of each 1000 deaths in 1905, distributed as follows:

| | |
|---|---|
| Variola | 5 |
| Measles | 380 |
| Scarlet fever | 130 |
| Whooping-cough | 300 |
| Diphtheria | 185 |
| | 1000 |

*Variola.*

*Annual Deaths per Million Living from Variola.*

| Decennium. | Deaths. |
|---|---|
| 1851–1860 | 221 |
| 1861–1870 | 152 |
| 1871–1880 | 230 |
| 1881–1890 | 44 |
| 1891–1900 | 13 |
| 1901–1905 (5 years) | 30 |

The rise to 30 per million in 1901–5 was due to the epidemic in 1903, which caused 2609 deaths, or 80 per million living. Although epidemics still from time to time occur, it will be seen that the mortality has lessened by 86 per cent. since 1851–1860. In the 1903 epidemic it was noticeable how many of the patients had been vaccinated once only—in infancy—and to completely stamp out smallpox re-vaccination is essential, but seeing the curious prejudice among the people of this country against anything official in nature, such a procedure is more likely to be carried out through the influence of medical men than by any fresh legislation.

*Measles.*

*Annual Deaths from Measles per Million Living.*

| Decennium. | Deaths. |
|---|---|
| 1851–1860 | 412 |
| 1861–1870 | 391 |
| 1871–1880 | 334 |
| 1881–1890 | 408 |
| 1891–1900 | 414 |
| 1901–1905 (5 years) | 326 |

* Presidential Address delivered before the Harveian Society.

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From 1881 to 1886 some deaths were attributed in each year's returns to German measles, but as this disease is not fatal the deaths were probably due to true measles, and are included in the above figures.

Measles, as may be seen, had a higher death-rate in the last complete decennium than ever before. This was owing to an unusually severe epidemic in 1896, the deaths being no less than 17,618, or an average of 572 per million living—the highest ever recorded except in 1887.

There is considerable lessening in the mortality in the five years after 1890, giving ground for hope that measles is decreasing in fatality. (In 1896 there was a further fall to 273 per million living.)

The mortality from measles is still greater than from any other infectious disease, and the ill-effects of the disease are much more than are shown by the number of deaths it produces, for many pulmonary, glandular, and catarrhal diseases date their onset from an attack of measles, and especially often is abdominal tuberculous disease preceded by it.

No less than 93 per cent. of measles deaths occur during the first five years of life, and most of all between the ages of two and three years; the great fatality before the school age militates against the idea that it results mainly from school attendance. Compulsory notification of measles has been advocated, but would do little good without isolation, and even with isolation the benefit would be much less than in the case of the other exanthema, measles being infectious before the rash appears. And to provide isolation accommodation for a disease of which the deaths alone numbered 17,706 in one year would need a great addition to our infectious hospitals, none of which in London, except the London Fever Hospital, admit measles.

Prompt closing of schools when a case of measles occurs might lessen its prevalence, but one reason for the great prevalence of measles is that parents, thinking it is certain to be contracted at some time, rather court than avoid a possibility of infection for their children. Medical men might well use their influence to discourage this custom, for a mild measles in one child may produce a long series of severe and even fatal measles in others.

### *Scarlet Fever.*

It was not until the year 1855 that scarlet fever was distinguished from diphtheria in the death-returns, and the first decade therefore for which statistics of scarlet fever are available is 1861–1870.

*Annual Deaths per Million Living from Scarlet Fever.*

| Decennium. | Deaths. |
|---|---|
| 1861–1870 . . . . | 890 |
| 1871–1880 . . . . | 649 |
| 1881–1890 . . . . | 312 |
| 1891–1900 . . . . | 158 |
| 1901–1905 (5 years) . . . | 126 |

The figures for the individual years in these decades show that there were wide variations in individual years corresponding to the occurrence of epidemics: thus, in 1863 the deaths rose to 1478 deaths per million; scarlet fever epidemics still occur, but their effect is quite insignificant as regards mortality compared with those of a few years back. Not once since 1893 has the mortality reached 200 per million, and the gradual and continuous decrease of scarlet fever is most remarkable—a decrease of 87 per cent. in 45 years, a greater decrease than in any other disease.

(P.S.—In 1906 the mortality fell still further to 101 per million.)

Is this diminution due to lessened prevalence of scarlet fever or to the disease becoming of milder type? It is not possible to ascertain its prevalence before notification of scarlet fever became compulsory, *i.e.* in 1890; even since that date its prevalence cannot be stated in figures for the whole country, notifications being made to the local authority. But the prevalence can be ascertained in the metropolitan area; and in the following table the notifications are given since 1891:

*Notifications of Scarlet Fever in the Metropolis 1891–1905.*

| | | | | | |
|---|---|---|---|---|---|
| 1891 | 11,398 | 1896 | 25,647 | 1901 | 18,381 |
| 1892 | 27,098 | 1897 | 22,848 | 1902 | 18,252 |
| 1893 | 36,901 | 1898 | 16,894 | 1903 | 12,531 |
| 1894 | 18,440 | 1899 | 18,089 | 1904 | 13,439 |
| 1895 | 19,757 | 1900 | 13,800 | 1905 | 19,461 |
| Average | 22,718 | | 19,455 | | 16,435 |

These figures show variations in individual years, but during the 15 years ending 1905 the prevalence of scarlet fever declined from an annual average of 22,718 to an annual average of 16,413—a decline of 28 per cent.

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The case fatality during the same period in the cases treated at the Metropolitan Asylums Board hospitals was as follows:

*Percentage of Deaths in Scarlet Fever Cases.*

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 1891 | 661 | . | 1896 | 4·29 | . | 1901 | 381 |
| 1892 | 728 | . | 1897 | 4·07 | . | 1902 | 345 |
| 1893 | 611 | . | 1898 | 4·12 | . | 1903 | 310 |
| 1894 | 592 | . | 1899 | 2·65 | . | 1904 | 337 |
| 1895 | 545 | . | 1900 | 2·97 | . | 1905 | 330 |
| Average | 6·28 | | | 3·62 | | | 3·4 |

It will be seen that from 1891 to 1905 the case fatality * fell from an average of 6·28 to an average of 3·4 per cent. annually—a reduction of 45·8 per cent. in the case fatality, as compared with a reduction of 28 per cent. in the prevalence of scarlet fever during the same period.

These figures apply only to the scarlet fever cases in London, and as far as case fatality goes only to that proportion (some 70 per cent.) of these cases which were treated in the Metropolitan Asylums Board hospitals, but they probably fairly represent the prevalence and case mortality in the country generally, and it therefore appears that the great decline in the mortality from scarlet fever in the last 45 years is in part due to lessened prevalence of scarlet fever, but in much greater part to the disease having taken on a much less severe type—a conclusion which, I think, will tally with the experience of all medical men who can compare scarlet fever now and twenty-five years ago. So great has been the change that the descriptions of scarlet fever in the text-books of some years back are scarcely applicable to scarlet fever as we now see it, its mildness being so extreme that there is often great difficulty in diagnosing its existence. Such gradual alteration of type is characteristic of most diseases which are on the decline and gives good hope of the continuance of the lessening dangers of scarlet fever.

Whether the diminution in scarlet fever mortality has been contributed to by the system of removal to special hospitals has been much questioned; personally I cannot doubt that the removal of a case of scarlet fever from private dwellings is of the greatest benefit to the patient whenever insufficient air-space and ventilation and good nursing is not there obtainable, and that removing a focus of infection from susceptible persons lessens the prevalence of the disease.

* If the case fatality is carried still further back it is found that it had fallen from over 10 per cent. in 1872 to 3 per cent. in 1906.

*Diphtheria.*

Before the year 1855 scarlet fever and diphtheria were returned as one disease—diphtheria. Distinctions were subsequently made which resulted eventually in affections which had before 1855 been grouped under one heading becoming tabulated under five separate heads—scarlet fever, diphtheria croup, membranous croup, laryngitis, and membranous laryngitis.

In 1894, disregarding its derivation from διφθερης, a membrane, the meaning attached to the term "diphtheria" was arbitrarily altered, and from that time onwards all cases in which the Kiebs-Loeffler bacillus was found were called "diphtheria," whether they showed membrane or not, and all other cases were excluded. Hence, the term "diphtheria" at the present day comprises many cases which would formerly not have been regarded as diphtheria, and excludes some cases which would have been so regarded.

One effect of this changed reading of the word "diphtheria" is that "croup" is now regarded as "diphtheria," and deaths which would at one time have been certified as from croup are now certified as from diphtheria in yearly increasing numbers. Thus an average of 133 deaths per million were certified as from "croup" in the years 1881–90, but only 17 per million in 1901–5; and no doubt the use of the term "croup" will die out entirely. To obtain a comparison of any value we must, therefore, take for comparison the sum of the deaths from diphtheria and from croup in each year, and not merely the deaths certified as from diphtheria.

*Annual Deaths per Million Living from Diphtheria and from Croup.*

| | Diphtheria. | Croup. | Total. |
|---|---|---|---|
| 1861–1870 | 171 | 219 | 390 |
| 1871–1880 | 112 | 140 | 262 |
| 1881–1890 | 153 | 133 | 286 |
| 1891–1900 | 263 | 51 | 314 |
| 1901–1905 | 204 | 17 | 221 |

The above table shows a decline of 43 per cent. in the deaths from diphtheria in the last 45 years. The decrease, however, is mainly in the last 5 years, and has not been long enough established

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to justify any conclusions as to a permanent decrease of diphtheria mortality. (P.S.—In 1906 the deaths from diphtheria and croup showed an increase over those of 1905.)

As in the cases of scarlet fever, some idea may be formed as to the cause of the diminished mortality by comparison of the prevalence—as shown by notifications—with the case fatality as shown by the cases treated in the Metropolitan Asylums Board hospitals.

*Notifications of Diphtheria in Metropolitan Area.*

| | | | |
|---|---|---|---|
| 1896 | 13,362 | 1901 | 11,968 |
| 1897 | 12,803 | 1902 | 10,538 |
| 1898 | 11,543 | 1903 | 7,582 |
| 1899 | 13,346 | 1904 | 7,073 |
| 1900 | 11,776 | 1905 | 6,358 |

This table from 1896 to 1905 (the term "diphtheria" being used in a different sense before 1896, comparisons are not carried further back), shows the rapid decrease in the number of cases notified as diphtheria. In the five years 1896–1900 the annual average was 12,565, in the five years 1901–1905 it was 8703, a decrease of 30 per cent.

*Percentage Case Fatality in Metropolitan Asylum Board Hospitals.*

| | | | |
|---|---|---|---|
| 1896 | 21·2 | 1901 | 11·1 |
| 1897 | 17·7 | 1902 | 11·0 |
| 1898 | 15·4 | 1903 | 9·7 |
| 1899 | 13·9 | 1904 | 10·0 |
| 1900 | 12·2 | 1905 | 8·3 |
| Average | 16·1 | Average | 10·1 |

This table shows the decreased case fatality from diphtheria in the quinquennium 1901–1905, as compared with 1896–1900, amounted to 37 per cent. (the figures apply only to notifications in London and to cases treated in Metropolitan Asylums Board hospitals). The diminution in the diphtheria mortality is due, it would therefore appear, in about equal proportions to lessened prevalence and to lessened severity of type of the disease.

In determining the effect of antitoxin treatment on the mortality, statistics give no assistance. The statistics usually put forward comparing the case mortality of diphtheria before and after the antitoxin treatment are misleading, for at the same time as that treatment came into vogue the connotation of the term "diphtheria" was radically changed. Since 1894 many slight cases of sore throat are included as diphtheria which would have been set aside as quinsy or tonsillitis, or catarrh, and would not have died under any treatment.

*Whooping-Cough.*

*Deaths Annually from Whooping-cough per Million Living.*

| Decennium. | Deaths. |
|---|---|
| 1851–1860 | 503 |
| 1861–1870 | 467 |
| 1871–1880 | 451 |
| 1881–1890 | 414 |
| 1891–1900 | 377 |
| 1901–1905 (5 years) | 300 |

The mortality from whooping-cough thus decreased by 40 per cent. in the last 55 years.

Whooping-cough, in addition to causing many deaths, is often the starting-point of respiratory affections and it is satisfactory that its lessening fatality is most marked in quite recent years—a diminution of 33 per cent. since 1880.

*Typhoid Fever*

was not distinguished from typhus fever in the Registrar's returns until 1868, and the first decennium in which its mortality is ascertainable is therefore 1871–1880.

*Annual Mortality from Typhoid Fever per Million Living.*

| Decennium. | Deaths. |
|---|---|
| 1871–1880 | 322 |
| 1881–1890 | 168 |
| 1891–1900 | 174 |
| 1901–1905 (5 years) | 112 |

There is thus a decline in the mortality of 68 per cent. in the last 35 years and the decline is going on with increasing rapidity, thus in

| | | | | |
|---|---|---|---|---|
| 1901 | deaths from typhoid were | | 155 | per million. |
| 1902 | ,, | ,, | 126 | ,, |
| 1903 | ,, | ,, | 100 | ,, |
| 1904 | ,, | ,, | 93 | ,, |
| 1905 | ,, | ,, | 89 | ,, |

(P.S.—In 1906 it was 92 per million.)

The following table gives side by side the notifications of typhoid fever in London and the death-

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rates in quinquennial periods since 1891 when notification became compulsory.

| Quinquennium. | Average notifications annually. | Average death rate per cent. in Metropolitan Asylums Board hospitals. |
|---|---|---|
| 1891–1895 | 3273 | 16·9 |
| 1896–1900 | 3612 | 16·5 |
| 1901–1905 | 2477 | 14·5 |

From this table it appears that while the prevalence of typhoid fever in the metropolis has decreased in the fifteen years 1891–1905 by 24 per cent., the case fatality has decreased in the same period by 14 per cent.

The period available for comparison is not very long, and as epidemics vary in severity too much stress is not to be laid on these results, but so far as they go they tend to show that the decreased mortality from typhoid is in part due to lessened severity and in part to diminished prevalence of the disease.

That the type of typhoid fever has considerably altered I pointed out in a paper read before this Society some years back; the profuse diarrhœa, high temperature, tendency to coma vigil, delirium, and the typhoid state, which gave so much difficulty in distinguishing between typhus and typhoid fever, were much more common twenty-five years ago than they are now, when constipation is as common as diarrhœa, the pyrexia often of low range and the mental condition clear throughout. The decline in the deaths from typhoid fever of some 68 per cent. between 1871 and 1905, and the increasingly rapid decline in the last five years, give reason to believe that a few years hence typhoid will be as extinct in England as is typhus fever.

### *Exanthemata Generally.*

The following table shows the mortality in decennial periods since 1850 from the infectious fevers taken together. Scarlet fever and diphtheria were grouped as one in the first decennial period, and typhoid and typhus fever were similarly coupled until 1870, but this does not affect the totals.

The number of deaths before compulsory notification, *i.e.* before 1874, are doubtless understated; the decrease in succeeding years is, therefore, even greater than these figures show. It will be seen that the total deaths per million annually from the exanthema have decreased by two thirds: while they accounted for 178 of every 1000 deaths in 1855 they accounted only for 62 of every 1000 in 1905.

*Deaths per Million annually from Exanthemata.*

| Decennium. | Variola. | Measles. | Scarlet fever. | Diphtheria. | Whooping-cough. | Typhus. | Typhoid. | Total. |
|---|---|---|---|---|---|---|---|---|
| 1851–1860 | 221 | 412 | 982 | | 503 | 913 | | 3233 |
| 1861–1870 | 152 | 391 | 890 | 390 | 467 | 886 | | 3176 |
| 1871–1880 | 230 | 334 | 649 | 261 | 451 | 58 | 322 | 2305 |
| 1881–1890 | 44 | 406 | 312 | 286 | 414 | 14 | 198 | 1674 |
| 1891–1900 | 13 | 414 | 158 | 314 | 377 | 5 | 174 | 1555 |
| 1901–1905* | 30 | 326 | 126 | 223 | 300 | 1 | 112 | 1118 |

* Five years.

The decline has been contributed to by all the exanthemata, least by measles; thus:

Scarlet fever has declined 87 per cent., variola 86 per cent., typhoid fever 68 per cent.; diphtheria 42 per cent., whooping-cough 40 per cent., and measles 20 per cent.

Typhus has vanished from the scene.

### *Influenza.*

The mortality statistics from influenza in each year since the epidemic began in 1889, up to 1905, have already been given when speaking of pneumonia; they show that it has never ceased to be prevalent since 1889 and is still with us.

The superintendent of statistics computes the deaths resulting directly from influenza in 1890, 1891, 1892, as 110,000, and these figures give no indications of the additional injury to health which indirectly resulted from influenza. The sudden increase of fatal pneumonia which occurred coincidently with the influenza has already been shown by figures, and every practitioner knows of the numerous respiratory nervous and cardiac affections that ensue from influenza without direct expression in the death returns.

The epidemic of 1847–1848 was even more severe, producing a mortality of 366 per million as against 317 per million in the 1890–1891 epidemic.

The contagiousness of influenza is now generally recognised, but scarcely sufficient importance is attached to the tendency of the influenza poison to lurk in bedding and textural substances generally.

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Here are some examples from among many I could give. Two students occupying adjoining rooms had influenza; when convalescent they went away and their lodgings were kept locked up till on their return from the country to their rooms. Each student at once got influenza again. A caretaker, the sole occupant of a large house, had severe influenza. She went into the country after this to recuperate; returned to her bedroom in London four weeks later to develop at once severe influenza again.

A medical friend went to a seaside resort for a short holiday; forty-eight hours later he got a very severe attack of influenza, and he then found the bedroom he lodged in had been but just before vacated by an influenza patient.

A family living in London had influenza in 1891. The children occupying two bedrooms had it, not the rest of the family. They went to their country house, returning in four months to town, when influenza at once attacked the children. This occurred in three successive years, and ceased after disinfection of the bedding.

Influenza has now prevailed for nearly twenty years and still prevails, and it caused 6951 deaths in 1905, and we hear constantly of persons having perhaps half a dozen attacks of it in a year.

I think its prevalence and its continual recurrence in individuals might be lessened if disinfection of bedding and sleeping rooms after attacks of influenza were more commonly practised.

### *Malignant Disease*

caused 60 of each 1000 deaths in 1905.

*Annual Mortality per Million Living from Malignant Disease.*

| Decennium. | Deaths. |
|---|---|
| 1851–1860 | 317 |
| 1861–1870 | 390 |
| 1871–1880 | 477 |
| 1881–1890 | 602 |
| 1891–1900 | 758 |
| 1901–1905 (5 years) | 861 |

There is, therefore, an increase of no less than 171 per cent. in the mortality from malignant disease during the last fifty-five years; possibly some of this increase is due to improved diagnosis, but no great part of it, for though sarcoma and cancer may be now more often differentiated that does not affect the above figures, which include the two together. And malignant disease that has become fatal is less likely to be wrongly diagnosed than many other affections. Unfortunately the mortality is still rapidly increasing. (P.S.—In 1906 there was a further increase to 918 per million.) Now, as always, the mortality is greatest in females, but this difference is lessening, for while the mortality from malignant disease has doubled in the female sex it has trebled amongst males.

### *Diseases Connected with Child-birth.*

These include all affections (sepsis, abortion flooding, etc.) incidental to child-bearing.

| Decennium. | Deaths annually per million women living. | Percentage mortality per registered births. |
|---|---|---|
| 1861–1870 | 312 | 4·69 |
| 1871–1880 | 343 | 4·75 |
| 1881–1890 | 335 | 4·73 |
| 1891–1900 | 295 | 5·09 |
| 1901–1905 (5 years) | 231 | 5·2 |

The decline in the number of deaths of women is of little value for comparison, as the birth-rate is decreasing. The registrar points out that the only comparison of value is between the puerperal deaths and the number of registered births, which the third column in the above table shows. The percentage of deaths is seen to be increasing; probably the same desire to limit families which leads to a lessening birth-rate brings also increased puerperal fatality.

### *Rheumatism and Gout.*

These together caused about 8 in each 1000 deaths in 1905; of these about one ninth were certified as from gout and the remaining two ninths as from rheumatism.

*Gout* is probably a more frequent cause of death than shown by the death returns, for it is much more frequently an indirect than a direct cause of death, and many deaths from gouty affections are certified without mention of their gouty origin.

### *Rheumatism.*

*Deaths Annually per Million Living from Rheumatism.*

| Decennium. | Deaths. |
|---|---|
| 1861–1870 | 115 |
| 1871–1880 | 140 |
| 1881–1890 | 127 |
| 1891–1900 | 115 |
| 1901–1905 (5 years) | 108 |

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In this table "rheumatism" "rheumatic gout" "rheumatism of heart" are included throughout, as rheumatic affections of heart were not separated in the Registrar's returns till 1900. It will be seen there is no material alteration in the fatality of rheumatism during the last forty-five years.

### *Diabetes.*

The death-rate from diabetes is ascertainable ever since 1850, and from that date to 1905 it has never ceased to increase: in 1850 the death-rate per million was 24 and in 1905 it was 92·9 per million living. The increase is too great and too constant to ascribe to greater precision in diagnosis, and at any rate in the last few years the accuracy of diagnosis has not increased, but the increased fatality of diabetes is as great as ever.

*Deaths Annually per Million Living from Diabetes.*

| Decennium. | Deaths. |
|---|---|
| 1851–1860 | 24 |
| 1861–1870 | 31 |
| 1871–1880 | 39 |
| 1881–1890 | 58 |
| 1891–1900 | 75 |
| 1901–1905 (5 years) | 89 |

(P.S.—In 1906 the mortality rose to 93 per million.)

The mortality from diabetes is still increasing year by year. In 1905 the death-rate had risen to 92·9, and in fifty-five years it has increased no less than 254 per cent. The deaths from diabetes number but a tenth of those from malignant disease, but the increase in the deaths from diabetes is even more rapid than that of malignant disease. This augumenting fatality of diabetes appears to prevail in all civilised countries. The mortality from diabetes is greater in males than in females—the reverse of cancer. The greatest mortality in both sexes is between sixty-five and seventy-five years of age, and diabetes, like nephritis, seems one of the causes of death likely to become more frequent as life becomes more often prolonged over the proverbial three score years and ten.

### *Alcoholism.*

*Annual Deaths per Million Living from Alcoholism.*

| Period of time. | Deaths. |
|---|---|
| 1858–1860 | 40·3 |
| 1861–1865 | 41·6 |
| 1866–1870 | 35·4 |
| 1871–1875 | 37·6 |
| 1876–1880 | 42·2 |
| 1881–1885 | 48·2 |
| 1886–1890 | 56·0 |
| 1891–1895 | 67·8 |
| 1896–1900 | 85·3 |
| 1901–1905 | 78·2 |

Each year increasing care is taken by the Registrar-General that only cases *directly* due to alcoholism shall be entered as such in the death returns, and many deaths that would formerly have been included under alcoholism are now classed among deaths from cirrhosis, hæmatemesis, etc.; the increase of deaths from alcoholism is, therefore, rather under- than over-estimated in the table given above.

It will be seen that the increase becomes greatest after 1889—the year when the influenza epidemic began; the death-rate from alcoholism rising from 55 per million in 1889 to 113 per million in 1900, a rise of 113 per cent., and one Chancellor of the Exchequer in his budget speech attributed the large balance at his disposal in part to the increased consumption of alcohol arising from its use in combating the depressing effects of influenza.

There is one satisfactory feature about the alcoholism returns—that the last five years ending 1905 show a decreasing mortality; thus in 1901 it was 96 per million; in 1902, 84 per million; in 1903, 76 per million; in 1904, 70 per million; and in 1905, 65 per million. (P.S.—It was 66 per million in 1906.)

The increase in deaths from cirrhosis has been already referred to

### *Tuberculosis.*

The decline in the mortality from tubercle of the abdomen by 37 per cent., of tubercle of meninges by 40 per cent., and of pulmonary tubercle by 55 per cent. has been already alluded to; there remain cases of tubercle affecting other parts, such as genito-urinary system, bones, joints, and lupus, which are grouped together by the Superintendent of Statistics as "tubercle of other organs." These show a slight increase, probably due to more careful diagnosis.

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*Annual Mortality per Million Living from "Tubercle of other Organs."*

| Decennium. | Deaths. |
|---|---|
| 1861–1870 | 118 |
| 1871–1880 | 103 |
| 1881–1890 | 163 |
| 1891–1900 | 186 |
| 1901–1905 (5 years) | 170 |

*Deaths Annually from all Forms of Tuberculosis per Million Living.*

| Decennium. | Deaths. |
|---|---|
| 1851–1860 | 345 |
| 1861–1870 | 324 |
| 1871–1880 | 287 |
| 1881–1890 | 242 |
| 1891–1900 | 201 |
| 1901–1905 (5 years) | 175 |

It will be seen there is a fairly uniform and continuous decline in mortality from tuberculosis, amounting to 42 per cent. in the fifty-five years ending 1905, and while tuberculosis caused 15·6 per cent. of deaths from all causes in the decennium 1851–1860 they caused only 11 per cent. in the decennium 1891–1900.

I shall not enter here into the question of infant mortality to which most of the groups of affections in the table contribute.

*Conclusions.*

Comparison of the table of 1000 deaths in 1855 and in 1905 show that—

(1) In 1905 as in 1855 diseases of the respiratory organs accounted for about one quarter of the total deaths; as already mentioned phthisis, however, has much declined in mortality, and pneumonia shows an increase.

(2) The apparent increase in deaths from disease of the circulatory organs and the decrease in those from diseases of the nervous system is due mainly to the transfer from the latter head to the former of all cases of apoplexy; the sum of deaths from circulatory and nervous disorders together in 1855 being 200, not very different from the sum of the two together—230—in 1905.

(3) The decreased deaths from tuberculosis is not shown in the table of 1000 deaths, being divided up among the respiratory, abdominal, and other affections, but it amounted to 42 per cent.

(4) The enormous decrease in the number of deaths from the exanthemata is seen by the fact that in 1905 they caused 75 deaths in each 1000 deaths as against 170 in 1855.

(5) The rise in mortality from malignant disease is seen in the 60 deaths in each 1000 deaths in 1905 as compared with 17 in 1855.

(6) The rise in mortality from diabetes is seen to be as marked as that from malignant disease.

(7) There is a greatly increased fatality from nephritis as shown by the rise from 16 to 30 per 1000 deaths between 1855 and 1905.

*May 11th*, 1908.

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A Dictionary of Medical Diagnosis: A Treatise on the Signs and Symptoms observed in Diseased Conditions. By Henry Lawrence McKisack, M.D., M.R.C.P.Lond. London: Ballière, Tindall and Cox.

In approaching a case of illness the observer is confronted with certain signs and symptoms, the significance of which he must be familiar with before he is able to form any intelligent conception of the morbid processes at work. It is, therefore, necessary to study carefully the language of signs as presented in the bodies of patients. Among the most fruitful methods of clinical teaching is that of considering fully the nature and origin of individual symptoms, observing in what respect the processes to which the symptom is attributed differ from those occurring in the healthy body. It is possible, by reviewing in this manner the subjective and objective symptoms, severally and collectively, presented by the patient, to indicate the nature and situation of the diseased condition; and it is precisely this comprehension of the nature of the process or condition, and not the mere labelling it with a name, which constitutes a rational diagnosis. The study of individual signs and symptoms of disease deserves more attention than has been devoted to the subject by writers. For some years the author has been in the habit of noting, for his own use and for teaching purposes, the significance of the various morbid phenomena, considered strictly from the observer's point of view. The views expressed above are taken from the author's preface to this volume, and accurately describe the nature of the contents of this remarkable work. Dr. McKisack has succeeded in placing within reach of his readers in an easily available form the information necessary for arriving at a rational diagnosis, based upon symptoms and signs commonly encountered in medical affections. This useful book will be most heartily welcomed by the present-day practitioner, whose difficulties are especially considered by the author and dealt with in a masterly manner indicative of a mature judgment ripened by a long and varied experience.

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# THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE, AND CLINICAL REVIEW.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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# SOME INTERESTING CASES OF CRANIAL SURGERY.

A Lecture delivered at the Polyclinic, London.

By HENRY CURTIS, B.S., F.R.C.S.,
Assistant Surgeon to the Metropolitan Hospital, Kingsland Road, N.E., and to the Branch Seamen's Hospital, Royal Albert Docks, E., and Senior Clinical Assistant in the Ear and Throat Departments of the London Hospital, E.

THE records of six patients under my care, which I have ventured to describe as interesting, comprise four groups:

(1) Two cases of depressed fracture of the skull, with extra- and intra-dural effusion, one of which was compound, and complicated with two separate and extensive fractures on the same side of the vertex.

(2) A case of gumma of the pericranium, and subjacent bone, occurring at the seat of repeated injury, and causing long-standing headache; operation; cure.

(3) A case of suppurative mastoiditis, and cerebral abscess, in which the symptoms were masked owing to thrombosis of the bulb of the jugular vein.

(4) And lastly, two cases of pneumococcic meningitis.

It is with the last two groups of cases that I want chiefly to occupy your attention.

The interest of the first case lies in the fact of the injury being in the left parietal region, causing aphasia and right-sided hemiplegia, clearing up after removing the pressure.

CASE 1.—*Depressed fracture of the skull in the left parietal region, with extra-dural hæmorrhage, and large sub-dural effusion, causing aphasia and right hemiplegia; trephining and cure.*

G., a Matabele boy, æt. 11½ to 12 years, was sent into the Memorial Hospital, Bulawayo, after

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an assault, of which the only detail known was that the injury was due to blows on the skull with a knobkerrie.

Arriving by train from an outlying district he was found to be suffering somewhat from shock, dazed, aphasic, and paralysed in the right arm and leg. The tongue could be protruded and kept in the median line.

T. 98·4° F., P. 72, R. 28.*

The patient remained in this condition until two and a quarter hours later, when, with dilated and equal pupils, there was twitching, chiefly of the right leg, and also of the left, the arms being unaffected.

Both knee-jerks were present, the left being brisk, the right exaggerated. T. 100° F., P. 78, R. 24. The boy now indicated the existence of pain on the right side of the nape of the neck, near the foramen magnum, but the tenderness on examination was only over the region of Broca's convolution, on the left side, and there were

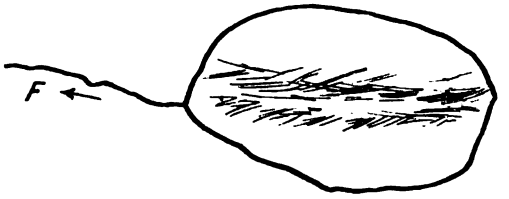


FIG. 1A.—F. Fissure leading out and down to base of skull.

obvious signs of a depressed fracture about the left parietal eminence.

*Operation, February 17th*, 1906.—With T. 98·4° F., P. 78, R. 28, and probably at least forty-eight hours after the injury, a boat-shaped depressed fracture of the left parietal eminence, measuring $1\frac{9}{16}$ in. by $\frac{15}{16}$ in., with a fissure leading to the base, was exposed (Fig. 1A).

After trephining and removing bone to the extent of 2 by $1\frac{1}{2}$ in., an extra-dural hæmorrhage was found, and, on clearing this away and incising the bulging dura mater, clear serum under pressure was evacuated, on removal of which the brain slowly began to pulsate (Fig. 1B).

An hour after the operation, T. 100·4° F., P. 120, R. 38.

February 18th: The next morning, T. 98° F., P. 102, R. 30.

In the evening the fits recurred, controlled by an injection of morphia, ♏ iss. Both knee-jerks practically abolished. T. 101·2° F., P. 106, R. 28.

In parenthesis it may be stated that any cause of depression, such as an operation, in tropical climates is apt to set up fever, probably from malaria, endemic especially in the natives, and the temperature chart in the case in question until discharge clearly showed the recurrence of malaria every seventh or eighth day, yielding to treatment by quinine.

February 19th: T. 100° F., P. 102, R. 30. No fits since the one dose of morphia last night. Pupils of medium size, not contracted. Right eyelids a little puffy (common in malaria, in my experience). Wound steadily healing. Brain pulsates normally. At 6 p.m. to-day the boy was

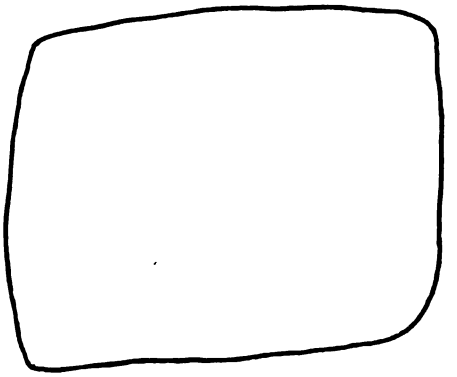

FIG. 1B.—Diagram of approximate size and shape of area of skull trephined and removed in treating above depressed fracture.

able to speak several words in English and Tebeli quite distinctly, for the first time since admission.

He can move the right arm and leg fairly well, but the hand grasp is feeble.

February 20th: The condition of arm and leg improves, and the boy speaks better, though slowly. Constant salivation. T. 99° F., P. 100, R. 22.

February 22nd: The œdema of the right eyelid remains, but has not increased. T. 98° F., P. 88, R. 22. Both legs can be flexed and extended quite normally. For the first time to-day the left knee-jerk present again; right knee-jerk still absent.

Right shoulder movement quite strong. Right hand given, when asked for, quite readily, but grasp not yet normal.

February 24th: Slight knee-jerk right side

* T. indicates, here and elsewhere, temperature; P., pulse-rate per minute; R., respiration-rate per minute.

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present; not much more marked on the left side. Still cannot grasp normally with right hand. Shoulder movements good.

April 10th: Steady improvement occurred, though not quite complete functional recovery to date, when the boy, temporarily discharged to attend a court of law some distance away, left, but never returned.

The following case contrasts with the last in that, whilst there were two extensive depressed fractures of the skull, there was practically no shock, and no paralysis or functional disturbance, though the injuries were severe enough to cause considerable effusion of bloodstained serum beneath the dura mater, and a hæmorrhage between it and the cranium.

Relief of the pressure by trephining was afforded in each case about the same time, some two and a half to three days, after the injury. Whilst in Case 1 the left side of the brain was compressed, causing aphasia and hemiplegia, in Case 2 the right side of the brain was affected, pressure being applied chiefly to the anterior part of the frontal lobe, which may partly explain the limited reaction.

Under the section relating to lumbar puncture a brief reference is made to its use as a means whereby a distinction can be made between extra- and intra-dural hæmorrhage: in cerebral laceration or sub-dural hæmorrhage the cerebro-spinal fluid is blood-stained, in extra-dural hæmorrhage the lumbar-puncture fluid is clear (Ballance).

CASE 2.—*Two compound depressed fractures in the same skull, with extra-dural hæmorrhage, and extensive sub-dural effusion compressing the brain; trephining in two places, etc.; cure.*—A Matabele boy, J., from the Morven Mine, Rhodesia, æt. about 15½ years, was admitted under my care on Tuesday, March 7th, 1905, at 9 a.m., with the history that on the Saturday (March 4th, three days previously) stones thrown up violently into the air, as the result of a dynamite explosion in the mine, had, in their descent, struck the boy on the head.

When admitted the boy was quite conscious, and beyond the fact of a slightly depressed pulse-rate and temperature, respectively 60 per minute and 96·8° F., there was no evidence of shock, the boy walking from the hand-cart in which he had arrived into the native ward.

There were two scalp wounds, long and deep, parallel with, and on the right side of the sagittal suture:

(1) In the right frontal region, extending as far forward as the frontal sinus, the mucous lining of which, indeed, was exposed in trephining later.

(2) Situated in the parieto-occipital, chiefly parietal, region.

Examination showed there to be an extensive and deeply-depressed fracture beneath the anterior of these two scalp wounds, and immediate operation was therefore decided upon.

*Operation.*—On reflecting the scalp in the right frontal region a long, but narrow, ovoid or boat-shaped depression was exposed, about 1¼ to 1½ in., its long axis being parallel with the median line of the skull (see Fig. 2, Diagram A).

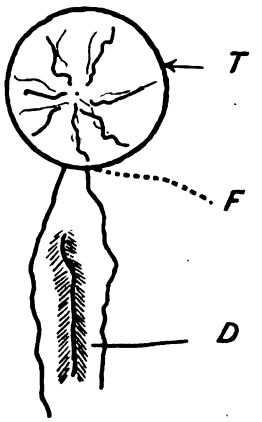


FIG. 2.—*Diagram A.* T. Trephine hole leading down to exposed mucous lining of right frontal sinus, including starred fracture in front of boat-shaped fracture. F. Fissure extending out and down to base of skull. D. Depressed boat-shaped fracture in right frontal region.

At the anterior end of the depressed fracture there was star-shaped fissuring, one of the fissures passing out and down and backward to the base.

A trephine was applied to the fissured fracture area, and on removal of the fragments the front of the trephine was seen to have extended as far forward as the right frontal sinus, the mucous lining of which was exposed.

The boat-shaped depression behind was then removed, and the exposed dura mater found to be bulging and cyst-like in appearance. On incising this a considerable quantity of blood-stained serum under pressure was evacuated, the brain beneath being markedly depressed, and not pulsating.

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The wounds in the dura and scalp were sutured, allowing for drainage.

The second incision, about 2½ in. in length, situated over the posterior half of the calvarium, had appeared, at first, a mere scalp wound, but on closer examination, and reflecting its edges, an extensive and wide boat-shaped depressed fracture was found, about twice the length and width of that seen anteriorly, with a fissure extending from its posterior margin backward, outward, and downward to the base, running through and almost bisecting another area of depressed bone, somewhat circular in shape, situated posteriorly

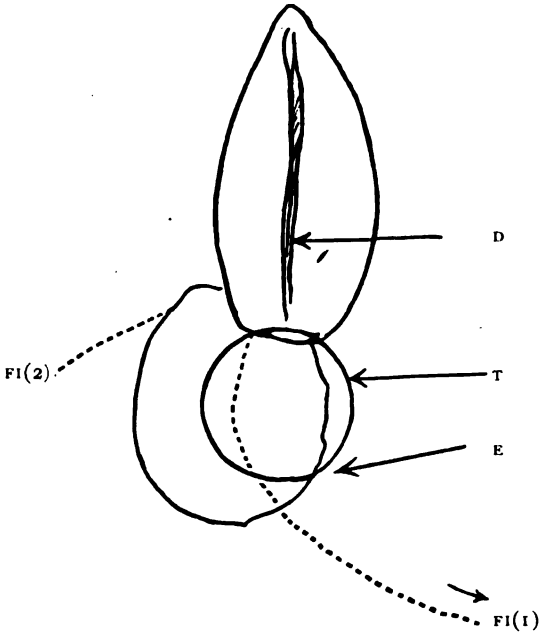


FIG. 2.—*Diagram B.* D. Boat-shaped depressed fracture, depressed chiefly along central line. T. Trephine hole placed just behind main depressed fracture, over a second area of fracture marked E. FI(1). Fissure extending out and down to base. FI(2). Fissure leading towards base.

and internally to the boat-shaped depression (see Fig. 2, Diagram B).

The trephine was applied posteriorly, so as to remove most of the depression behind the main boat-shaped area, and then the remaining depressed bone elevated and removed as usual. An extensive area of extra-dural blood-clot was exposed, and as much as possible was cleared out. The wound was sutured, allowing for drainage.

During the operation liquor strychninæ, up to ♏ xv, and Tr. digitalis, ♏ x, required to be injected, followed up after the operation by rectal injection of brandy, the pulse being feeble and slow, the respiration natural.

The boy was discharged forty-six days after admission with the wounds healed, there being no evidence of mental disturbance.

Recovery, considering the presence of two such extensive fractures on one side of the skull, if it does not constitute a record, is remarkable, especially if the septic state of the wounds—in the case of a Kaffir's scalp—after three days' exposure to germs of every kind, be remembered.

The third case of the series illustrates the facts (1) that though the spirochæte of syphilis produces a generalised infection, it shows a well-marked tendency to settle in certain tissues and organs, and amongst bones, notably the skull; and (2) that exposure of these tissues or organs to injury, by creating an area of still lower vitality than usual, greatly increases their liability to such infection.

This is comparable with the experimental fact that no effect may be produced on inoculating an animal with a staphylococcus derived from a virulent lesion in man until an area is met with, or created, where the resistance has been lowered, such as the site of a fracture.

Another instance of this was afforded in the lecture on "Liver Abscesses" I delivered at the Polyclinic last year:

Dysentery led to amœbic abscesses of the liver rupturing through the lung, there being also an associated appendix abscess. As the result of free drainage, recovery was progressing when the most profuse discharge set in, the prognosis becoming almost hopeless. Potassium iodide, pushed to 35 gr., four times a day, for twenty-nine days, before iodism occurred, resulted in complete and permanent recovery, by absorbing the gummata which were complicating the liver abscesses.

We are once more reminded that in making a diagnosis the patient's occupation should always be borne in mind.

In the two cases just alluded to the men had been in the Army; and specific trouble may aptly, if a little unkindly, be included amongst the "Diseases of Occupation."

CASE 3.—*Severe headache, with localised tenderness over scarred left parietal region; trephining and removal of large gumma of pericranium and*

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*eroded bone beneath; cure.*—A white man, A. B—, æt. 38 years, who had been in the Mounted Police, was admitted under my care, February 20th, 1905, for persistent severe headache of several years' duration, and tenderness of the scalp, both referred to the left parietal region, where, on shaving, there were seen to be numerous linear scars, the result of blows on the skull received at various times. The scars together formed a figure like the letter Z, the lower bar of that letter forming a line 1¾ in. long, and running obliquely upward and backward, so as to lie about 1¼ in. in front of the left parietal eminence (Fig. 3). There was acute tenderness, especially marked at two points, located to the line of the scar, and at the lower anterior end of this a hard boss, more than ¾ in. in diameter, and a depression could be made out, apparently in the skull itself. The history of several injuries, and the presence of extensive scarring, under these circumstances, suggested the condition to be due to localised periostitis, with, perhaps, depression or new formation on the internal surface of the bone, and thickening of the meninges.

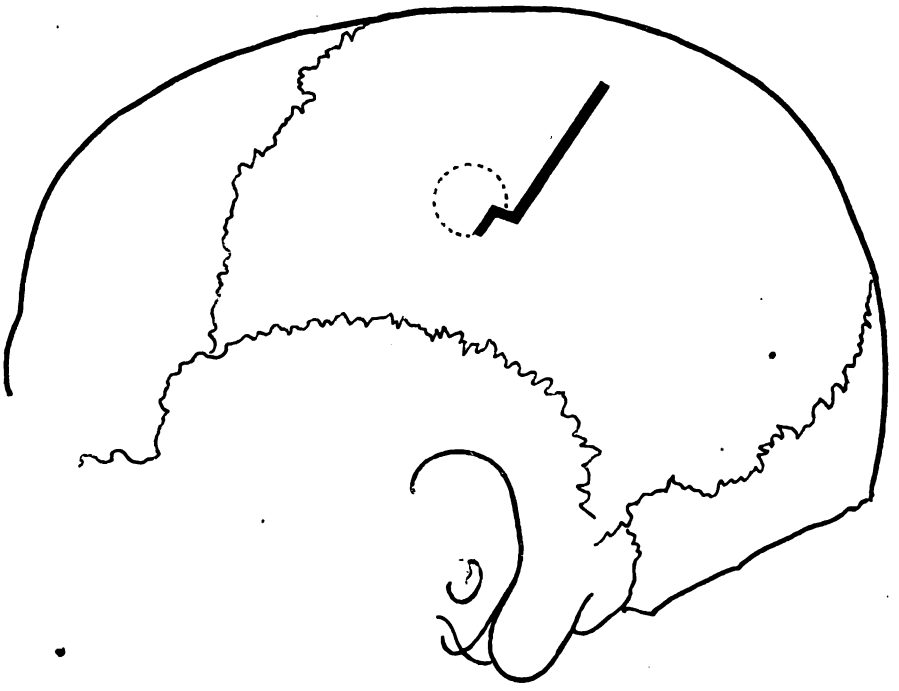

Fig. 3.

Exploration, being agreed to, was performed five days after admission, trional and morphia being, meanwhile, required occasionally for the relief of insomnia or pain.

*Operation, February 25th*, 1905.—On turning down a flap of scalp, which included the linear scars already noted in the skin, the large circular gumma now exhibited (Fig. 4), and, before shrinkage in formalin, the size and shape generally of an adult human patella, was seen involving the pericranium, the deep aspect of which was roughened and exhibited numerous punctiform apertures. The bone beneath was worm-eaten or eroded superficially, and as it was trephined I am able to show you the specimen (Fig. 5).

The bone corresponding to the gumma, of great thickness and density from syphilitic sclerosis, was freely removed, with a view to ascertaining the condition of the dura mater beneath. This appeared to be congested and considerably thickened, but with the removal of the lesion in the pericranium and treatment by iodide it was hoped that complete relief would be afforded, and the dura was therefore left intact.

February 26th: On the day following the operation there was, for the first time for several

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years, no headache, merely the dull pain from operative interference. Potassium iodide was ordered.

March 2nd: On the fourth day after operation the patient sat up, and wanted to smoke the next day.

March 11th: There has been no headache since the operation, and the dull pain due to operation passed away some days ago.

March 14th: Patient discharged to-day, quite free from pain and tenderness, and there was no subsequent recrudescence of symptoms whilst under observation for the next fifteen months.

The lessons to be learnt from this case are: (1) In persistent headache we should always think of a

FIG. 4.—Case 3. Gumma of pericranium, deep aspect.

local cause; and (2), where there is an obvious bone lesion, ever keep in mind the possibility of syphilis, even when there is evidence of an old injury, which so often determines the location of a gumma, by providing a *locus resistentiæ minoris*.

With the long history and advanced changes in the bone it is doubtful whether iodides alone would have effected a cure, and if a cure, a permanent one, pain often recurring locally on omitting treatment. It is certain that operative measures combined with iodide produced the most rapid and permanent effect in the present instance. Necrosis of the outer table of the skull, leading to the formation of a small sequestrum with suppuration around, has, in cases not operated on, resulted in perforation of the skull, and fatal meningitis or cerebral abscess (See Fig. 84, vol. i, 'Treves' Surgery').

Coming now to the last two groups of cases, the conditions present will repay careful study, meningitis, localised or diffuse, lateral sinus thrombosis, and abscess, cerebral or cerebellar, forming, of course, the three chief complications which may supervene upon middle-ear and mastoid disease.

A brief recital of the cardinal symptoms of these intra-cranial complications, and a consideration of the reliability of some of them, may be useful, and render more interesting the perusal of the cases here recorded.

We can then see how far these have conformed

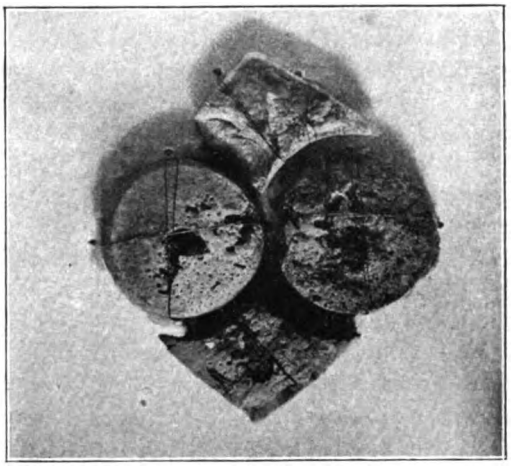

FIG. 5.—Case 3. The portion of superficially eroded and much thickened parietal bone removed at the operation.

to the type, and begin to appreciate what judgment and protracted observation are necessary for their correct diagnosis in the presence of complications, which may completely obscure the primary and essential lesion

*Meningitis.*—Typically, there is a sudden, or more often, insidious onset, with headache, irritability, photophobia, delirium, and, at a late stage, the characteristic "meningitic cry," together with retraction of the head, and some stiffness or rigidity of the neck muscles, where the posterior fossa is involved. There may be more or less transient squint and other evidence of implication of the cranial nerves; also, generally, optic neuritis; but in the rapidly fatal cases there may be no time for its development. There is persistent fever, and

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rapid, irregular pulse. At the commencement there may be rigors and vomiting. The abdomen is sometimes hollowed out, and the *tâche cérébrale* obtained.

Then there is the further test, very valuable when present—and it is almost always present—known to you as—

*Kernig's sign.*—With the patient propped up, or lying on the side, so that the thighs are bent on the trunk, there is contraction of the flexor muscles when attempts are made to fully extend the knees.

It may also be present in cerebral abscess, but is more generally absent (Potts).

*The value of lumbar puncture*, as an aid in diagnosis, will subsequently be discussed.

*Lateral sinus thrombosis* is now often spoken of as "sigmoid thrombosis," since the ᔓ or "sigmoid" portion is that most generally affected (see Fig. 7). The symptoms, resembling those of septic thrombosis elsewhere, are characteristic, consisting of rigors, accompanied by sudden sharp rises in the temperature, rapidly subsiding, only to ascend as sharply again, with the onset of another rigor. The rigors are frequent, and are sometimes preceded by great thirst and sweats. The pulse is very rapid and weak. There is great restlessness, and often delirium, and pain and tenderness localised to the side of the head or neck, or to both. Optic neuritis is present in 50 per cent. of the cases (Tod).*

Too great stress can hardly be attached to the onset of rigors, especially in the presence of chronic otorrhœa, clearly indicating under such circumstances implication of the lateral sinus. Case 4 affords an illustration of the ambiguity arising, however, when the clot is low down, and practically at the commencement of the jugular vein.

High leucocytosis, with a high percentage of polymorphonuclears, illustrated in Case 6, occurs, as usual elsewhere, in this septic condition.

In Case 6, it is to be noted, sigmoid thrombosis resulted in a case of pneumococcic infection from the tonsils, and almost certainly formed the direct route of invasion of the meninges. The case is further remarkable by reason of the absence of rigors throughout, in spite of the thrombosis having occurred very early in the course of the disease.

* 'Diseases of the Ear,' 1907.

*Cerebral abscess.*—The classical symptoms of this condition are:

(1) Headache, more or less localised, associated with mental and physical lethargy and apathy. The patient at first can be roused, and then gives intelligent answers to inquiries; but increasing drowsiness sets in, and finally coma.

(2) A subnormal temperature.

(3) A slow pulse; and—

(4) Vomiting.

With these signs and symptoms, if there is a history of chronic otorrhœa, and especially if there is recurrent vomiting of "cerebral type," *i. e.*, sudden, gushing, and without effort, independently of food, one may fairly safely diagnose cerebral abscess, which is rendered still more probable in the presence of optic neuritis.

Optic neuritis is, however, generally only seen at a comparatively late stage, owing to its slow development; some say only when the abscess is of large size and very chronic.

It is evidence of intra-cranial pressure. Even in cerebral tumour, as Ormond has shown,* out of 83 cases, at Guy's Hospital, it was definitely stated that in 13 optic neuritis was absent. Of these 13 cases 6 were situated at the base of the brain and directly pressed on the optic tracts or nerves.

Therefore, as a sign of intra-cranial pressure, though the presence of optic neuritis is helpful, its absence is of no value in excluding cerebral or cerebellar abscess. Case 4 will serve to emphasise this point, as also the a-typical state of the temperature and pulse, in the presence of complications, such as thrombosis, or meningitis.

Of irritative or paralytic symptoms, aphasia, motor or sensory, hemiplegia, oculo-motor or facial nerve palsy, etc., we need not here speak, as their absence is of no value, and though of use as localising symptoms when present, too often they arise at so late a period as to preclude much hope of operative relief.

Kernig's sign, as stated, may occasionally be present.

*Of the more recent aids to diagnosis, lumbar puncture* does not help, the fluid evacuated in uncomplicated abscess being free from pus (see later).

*The remaining test, the presence of high leuco-*

* 'Med. Chir. Trans.,' 1906.

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*cytosis*, with an excess of polymorphonuclears, is of real value when present.

Potts,* of Philadelphia, indeed, places great reliance upon this sign:

"The presence of 80 per cent., or more, of polymorphonuclears, in his experience, has always shown sepsis, not necessarily of the brain. It may be septic thrombosis, or other general septic process [see Case 6], but its presence has never failed to indicate sepsis. Its absence does not contra-indicate sepsis, as shown in cases that have been reported in which, with a high degree of pyæmia, the polymorphonuclear percentage may be as low as 70. [This agrees with our own Case 4.] But given a group of symptoms pointing to an infection the presence of a high polymorphonuclear percentage will in most cases be the deciding factor."

We see, then, that in typical cases the diagnosis presents no difficulty, but, as Potts says, no symptom is pathognomonic, and no group of symptoms, even, found to be infallible. Where the symptoms are masked by the presence of other intra-cranial complications, such as meningitis or lateral sinus pyæmia, it may be impossible to correctly appraise the value of the symptoms. Of 17 cases of cerebral abscess occurring in German hospitals during two years, referred to by Martius,† not one was recognised during life, and such a list of unrecognised abscesses could, unfortunately, readily be extended without much research.

Alluding to Dr. James Collier's important paper‡ dealing with the false localising symptoms of intra-cranial tumours, Dr. Beevor§ has recently remarked that these are such as to "mislead the unwary—aye, and the wary, too." These words may, with equal truth, be applied to the diagnosis of cerebral abscess.

*Cerebellar abscess.*—In addition to the symptoms accompanying cerebral abscess there may be sudden attacks of severe giddiness, with relaxation of the limbs, so that the patient may collapse in the street, half unconscious.

But for an account of some of the difficulties in diagnosing cerebellar abscess let me refer you to an excellent lecture by Wilfred Trotter,* who emphasises once more the importance of operation immediately the condition is diagnosed, by reason of the great danger of sudden respiratory failure, owing to pressure affecting the centre in the medulla. In the following week, Carter,† of Leicester, recorded a most interesting case of the kind, recovery following the opening of a cerebellar abscess, though artificial respiration had to be carried on throughout that operation for nearly half an hour, owing to the onset of respiratory failure at the very commencement of the anæsthetic (chloroform).

Respiratory failure preceded cardiac, in the final issue of the case of cerebral abscess following mastoiditis which we are now to consider.

CASE 4.—*A case of suppurative mastoiditis, leading by direct continuity to a large thick-walled abscess greatly distending the left temporo-sphenoidal lobe; absence of optic neuritis, and masking of other symptoms by thrombosis of the bulb of the jugular vein.*—The patient, a Polish Jewess, æt. 8 years, had long suffered from left otitis media, and was sent by Dr. M. Corner, of Mile End Road, to the Metropolitan Hospital for left-sided otorrhœa, and some tenderness over the mastoid. The child, who did not look very ill, also complained of frontal headache, localised to just above the glabella, and vomiting. She vomited in the Casualty Department suddenly, without retching or effort, evacuating a large quantity of food, etc.

The house-surgeon, Mr. Ilott, at once recognising the gravity of the conditions present, admitted the child the same day, September 3rd, 1907, when the temperature was 100° F., pulse 132, and respiration 24 per minute.

From the character of the vomiting chiefly, he suggested the provisional diagnosis of cerebral abscess.

On examination it was found that the otorrhœa was not very profuse or very fœtid; the left ear did not stand out, there was no redness, no tenderness, and very slight, if any, œdema over the mastoid. Even on firm pressure there was no tenderness either over the mastoid or at any spot on the left side of the skull.

The child, however, hardly understood English, and investigation therefore was not easy.

Ophthalmoscopic examination on several occasions, on admission and subsequently, negatived optic neuritis.

The scalp was covered with scabs and in a filthy condition; efforts were at once made to render it as aseptic as possible.

* 'Amer. Journ. Med. Sci.,' July, 1907, "Difficulties in the Diagnosis of Brain Abscess."

† 'Lancet,' January 5th, 1895, p. 12.

‡ 'Brain,' vol. xxvii, p. 490.

§ 'Trans. Med. Soc.,' London, 1907, "Diagnosis and Localisation of Intra-cranial Tumours."

* 'Brit. Med. Journ.,' March 14th, 1908.

† *Ibid.*, March 21st, 1908.

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As to the mental condition the child was quite conscious, rather silly and irritable, but not more so than any other Jewish child out of sorts. In the ward she wanted everything in a hurry, and screamed out quickly when asking for things.

The child did not complain of headache after admission until the same night, when, with a temperature of 103·6°, for the first time after coming in she vomited once.

The evidence for cerebral abscess was not strong; there was but little headache, there was no drowsiness, the temperature ranged from 100° to 103° F., the pulse was rapid, 110 per minute, and localised pain and optic neuritis were absent. In view of appeared in every way normal, and not to be thrombosed.

In the presence of definite and extensive mastoid disease it was felt unwise to explore the temporo-sphenoidal lobe through the area of dura mater exposed in the wound: partly because of the risk of conveying infection, if not already present; and partly because, as is well known, the drainage of the mastoid not infrequently clears up "cerebral" symptoms in children, which have been associated with mastoiditis, but are due to disease confined purely to the mastoid, etc., and are probably a manifestation of sapræmia attributable to small doses of toxines, from pus pent up in the antrum,

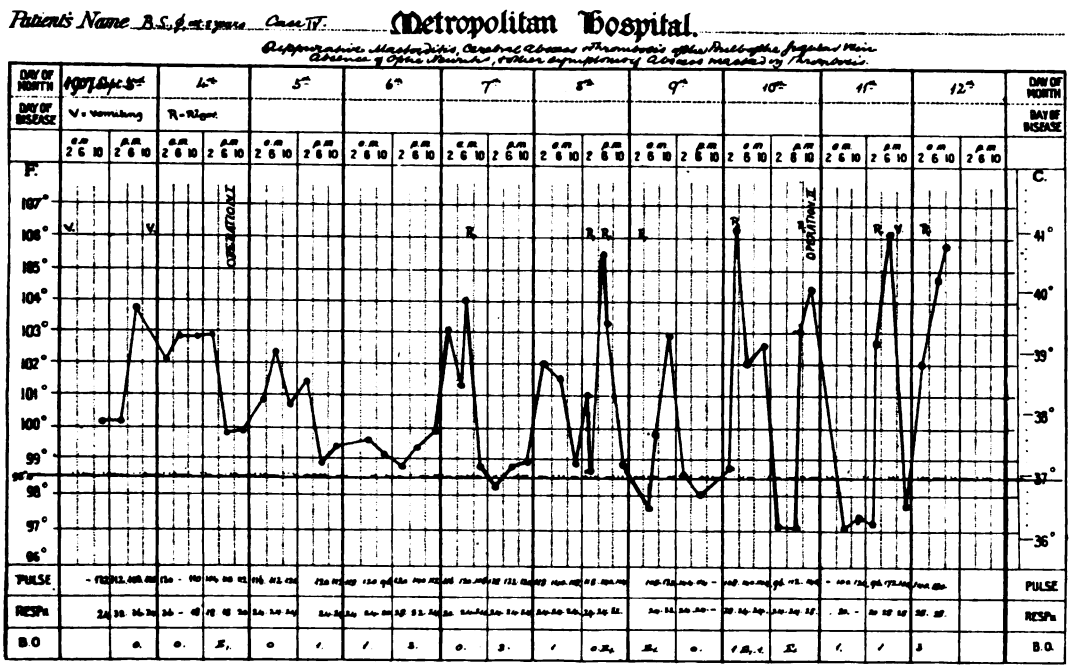


FIG. 6.—Temperature chart, Case 4. Suppurative mastoiditis, cerebral abscess, and thrombosis of the bulb of the jugular vein. The symptoms of cerebral abscess were masked by those due to the thrombosis. V = vomiting; R = rigor.

the chronic otorrhœa it was decided to explore the mastoid as a preliminary measure.

*Operation* 1: *Left mastoidectomy.*—The "complete mastoid operation" was therefore performed on the day after admission, a quantity of fœtid *débris* and a small quantity of pus being evacuated from the left mastoid antrum, which was deep-seated, extending inwards and forwards for a considerable distance, and, like the floor of the tympanic cavity, covered with granulations. In opening up the antrum, the dura mater just behind and above it was exposed, and appeared quite normal, there being no congestion nor the slightest evidence of tension.

The sigmoid portion of the lateral sinus was also exposed behind and below the antrum, and being forced into the neighbouring cerebral circulation.

B. H. Potts* records a case in point, with "symptoms of cerebral abscess, quite pronounced, which cleared up nicely, under the radical mastoid operation, even with a pulse of 60, marked changes in the optic discs, sluggishness and vertigo, vomiting and headache."

Meanwhile there had been only one attack of vomiting, namely, on the night of admission, not since, even after the operation. On recovering from the anæsthetic the headache was found to be greatly relieved, and the child took food well.

On the morning after the operation the patient was drowsy and disinclined for food. The tempera-

* *Ibid.*

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ture—but not the pulse and respiration-rates—fell through the day, so that by 6 p.m. it was 98·8° F. The patient slept well that night.

Three days later there was still no vomiting, but rigors, with the swinging temperature typical of sinus thrombosis, set in and continued for five days, until the day before death, which occurred eight days after mastoidectomy, or nine days after admission.

With the rigors there was marked headache and restlessness, but in the intervals the child was quite bright, played with its toys, and ate well. Drowsiness was only recorded twice, once the morning after mastoidectomy, and once the night before the second exposure of the sinus.

During my absence from town for three days the case was kindly seen once by a colleague, and the general and mental condition were so good as to seem to negative urgent exploration.

*Operation 2.*—On the fourth night of the rigors, when the leucocyte count was only 10,000 per cubic mm., the temperature being 104° F., pulse 140 per minute, a further exposure was made of the entire sigmoid sinus. It again presented a normal appearance, and there was no bulging of the dura mater, which looked quite healthy. The left internal jugular vein, from surface examination of the neck, appeared unaffected.

After this operation the child vomited once, but slept well all night, and then seemed better.

The child seemed quite bright till 3 p.m. the next day, when she became very thirsty, and a rigor occurred; temperature = 103° F., rising by 6 p.m. to 106·2° F., when another rigor occurred. She vomited once during the evening.

There was much pain over the left brow and eye, and a good deal of blinking movements of both eyes.

The child, at first restless, slept well until 4 a.m. the next day, when a slight rigor occurred; temperature = 104° F. Sharp vomiting ensued after a little tea. Marked headache at 6 a.m., becoming more and more severe, the child remaining quite sensible. At 8 a.m. the patient was quite quiet, but conscious. There was no irritability or further rigor. Pupils of medium dilatation. The respiration became very slow and sighing, this being very audible. It finally ceased, and death occurred at 9 a.m., the temperature being 105·8° F., the pulse remaining of very good quality up to the end, as it had almost throughout the disease.

Let us now read the notes of the post-mortem findings.

*Autopsy.*—By Dr. E. H. Shaw, Pathologist, etc., to the Metropolitan Hospital. (Examination of head only allowed.)

*Dura mater.*—Roughened opposite small opening in roof of left middle ear, but no pus present.

Leading from this point is a small track of sloughy material which is adherent to the brain; this, again, leads into the left temporo-sphenoidal lobe of the brain, which is swollen and contains a large abscess about two inches in diameter. The abscess contains a broken-down mass of brain tissue and pus (thick, dirty green and blood-stained pus, not notably fœtid). It has a well-marked wall of a brownish material about $\frac{1}{16}$ in. thick.

The remainder of the brain and cerebellum are natural in appearance.

The pia-arachnoid at the base is slightly thickened and white, but there is no meningitis away from the immediate neighbourhood of the above-mentioned track.

The petrous bone is hollowed out almost to its apex, the cavity containing a soft, discoloured mass of granulation tissue; the cavity is in front of the internal ear, and extends right up to the genu of the canal of the internal carotid artery.

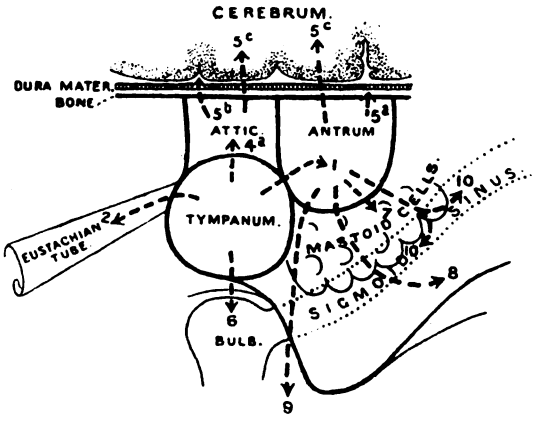


FIG. 7.—To illustrate routes of infection in Case 4. (From Tod's 'Diseases of the Ear,' Oxford Medical Press, by permission.)

*The left lateral sinus.*—Low down in the terminal part of the sinus and commencement of the left internal jugular vein is a small amount of pus and soft blood-clot, free and non-adherent to the wall. No more pus can be found lower down. (There was no pus outside the vein or sinus, the internal and external walls of the latter presenting a healthy appearance, and no evidence of any parietal clot.)

*Bacteriological examination.*—The pus was found to contain Gram-positive, strepto- and staphylococci, and long narrow bacilli. There was no growth in broth, nor on gelatine or agar.

*Remarks.*—The results of this autopsy, limited though it was, show that there was a small infected thrombus, practically confined to the bulb of the jugular vein, just at the termination of the sigmoid sinus, impossible to detect when the lateral sinus was fully exposed in the ordinary way.

In the corresponding temporo-sphenoidal lobe was a large thick-walled abscess, in direct continuity with the subjacent mastoid antrum, with no

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evidence (such as lepto-meningitis, by leakage into the subdural space, or by rupture into the ventricles) of having infected the sigmoid sinus.

Thrombosis of the lateral sinus commonly occurs in the sigmoid portion, but, as Tod* says: "Occasionally the bulb of the jugular vein becomes infected through the floor of the tympanic cavity."

This space, in the present instance, was noted to be very extensive and full of *débris*, unhealthy granulations, and pus, and in the light of the operative and post-mortem findings we may conclude that infection occurred along this route, a complication independent of the cerebral abscess, which had been previously infected from the suppurating mastoid antrum, supervening on the chronic otitis media.

It is obvious that discovery of this condition of the jugular when the rigors first set in, and ligature of the vein in the neck—the appropriate treatment when, in spite of rigors, the sigmoid sinus itself is not found thrombosed—would not have sufficed to save the child, the large cerebral abscess having evidently been present before admission.

The recollection that such a collection of pus could so easily have been tapped through the area of dura mater exposed at the mastoidectomy operation is not pleasant, but one has already stated the grounds for not exploring then, a decision which in this case of abscess, with anomalous symptoms resulting from the independent thrombosis, proved so unfortunate, though I think you must agree that, under any treatment, the prognosis was, from the first, practically hopeless, for the temperature chart indicated that pyæmia from jugular vein infection was also impending on admission. Had a more complete autopsy been permitted we should probably have found evidence of infection in lungs, liver, and spleen.

To prevent a future case sharing the same fate let us consider the ideal treatment, supposing recovery still to be possible. Under similar circumstances one would drain the antrum, and with the least suspicion of abscess, even in the absence of optic neuritis, and in the presence of fever and a rapid pulse, explore for cerebral or cerebellar abscess, either through the same skin wound, or through a separate incision, the former generally being unattended with much risk, after packing the laid-open mastoid antrum.

As a measure of precaution, even in the absence of rigors, where a cerebral abscess was thus discovered associated with fever and a rapid pulse, it would be wise to explore the lateral sinus and treat it, or ligature the jugular vein, as indicated hereafter.

The majority of cases will not be rendered so difficult as was the one under consideration, as, at any rate, where the abscess is of large size and has existed some little time, optic neuritis, though by no means invariable, as we have seen, may generally be expected; and in the few where there is the further complication of bulb thrombosis, there is the greatly-added probability of this sign being present, the incidence of optic neuritis in lateral sinus thrombosis being, as already stated, 50 per cent. In addition, of course, there would be the characteristic rigors. Case 6, however, serves to illustrate the fact that, exceptionally, these may be absent throughout in a marked case of thrombosis.

Having drained both the mastoid and brain, the onset of rigors would at once demand exploration of the sigmoid sinus (if not already performed), plugging it above and below any clot found, before evacuating it, so as to obviate general infection. Preliminary puncture of the vein, with the view of testing whether the sinus is thrombosed, is to be avoided, as hæmorrhage naturally follows if the puncture happens to be proximal to the clot; and it is likely therefore to be misleading. It is still more objectionable for another reason, since such a manœuvre tends to detach any parietal clot forming, and so disseminate infection into the general circulation.

In the comparatively infrequent event of finding the lateral sinus healthy, when rigors have once set in, the bulb of the jugular may be relied upon to be thrombosed, requiring exposure of that vein in the neck, the proximal end of the divided jugular being brought out of the upper angle of the wound, so that the thrombus can be dealt with without risk of disintegrated particles escaping into the general circulation.

The great lesson to be learnt from the case under consideration is that an important sign of intra-cranial pressure, such as optic neuritis, may really be absent, and some of the cardinal signs of cerebral abscess masked, by reason of some complication, such as meningitis, or sinus thrombosis; and where rigors occur one must not be deluded into believing thrombosis is absent because the sigmoid portion, even when thoroughly exposed, appears, and actually may be, healthy. Under these circumstances the jugular vein must be exposed and divided, its proximal end brought out of the wound and treated as I have already indicated.

As to *the occurrence of cerebral abscess in children*, Hott,* at the American Pediatric Society, stated that cerebral abscess in children is rare in the first four years of life, but is more common later on. He reported the youngest case on record in a child, æt. 6 weeks, two ounces of pus being evacuated; also one case at three months; of cerebellar abscess, one of his cases occurred at three months, and another at nine months.

* *Ibid.*

* 'Pediatrics,' June 1st, 1897; see also 'Lancet,' p. 212, July 24th, 1897.

*May* 18th, 1908.

(*To be concluded.*)

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# WITH MR. SHERREN IN THE WARDS OF THE LONDON HOSPITAL.

*Two Cases Illustrating the Treatment of Ulcerative Colitis.*

THIS patient, a man, æt 36 years, has suffered from dysentery, which he contracted in India eighteen months ago. He has been in this hospital three times, and although he has improved a little under medical treatment, at no time was the number of daily motions less than three, and at no time did he pass a normal one.

When I operated upon him he was passing from six to ten typical dysenteric stools daily, and the sigmoidoscope revealed ulceration of the sigmoid, the rectum being healthy. Four weeks ago I performed appendicostomy. He is now apparently cured; his bowels are only open once a day, and the motion passed is normal in colour and consistency, and there is no trace of blood, sloughs, or pus.

The operation of appendicostomy is simple: the appendix is sought for through the usual "muscle-splitting" opening in the right iliac fossa, and is brought out through the wound after dividing any necessary adhesions, and in some cases the meso-appendix. The cæcum is then united to the parietal peritoneum of the anterior abdominal wall by fine silk stitches, one above and one below the base of the appendix, and the muscles brought together around it and the skin sutured. As an extra precaution a sterile safety-pin may be passed through the meso-appendix. The appendix should not be cut off until the wound is healed. In eight or ten days it is removed flush with the abdominal wall and irrigation started. No difficulty is experienced with the washing out; the lumen of the appendix will admit, in most cases, a No. 12 English catheter. Irrigation should be done twice daily with plain sterile water or boric lotion. It will rarely be necessary to use chemicals; if it is, a solution of ¼ to ½ per cent. protargol is the most efficient. The washes are to be continued twice daily until no blood, pus, or sloughs are present. They should then be discontinued and the condition of the stools carefully watched, if they are normal no further washes are required. The patient should be kept under close observation for at least a fortnight after this.

It sometimes happens that appendicostomy fails to cure or even to relieve the condition. This, the patient in the next bed, a boy, æt. 19 years, illustrates. He was taken ill suddenly last May with abdominal pain, vomiting, and diarrhœa; this latter continued, and in July appendicostomy was performed, and he was irrigated with various solutions. This gave him no relief and his condition became steadily worse. He was admitted under my care on September 18th, weighing 5 st. 3 lb. He had pustular eczema of the face and neck, bedsores over the sacrum and right great trochanter, and was passing daily from ten to twelve offensive liquid stools containing sloughs, blood, and pus. Immediate operation seemed inadvisable, so I put him on daily intra-venous saline and glucose injections. This improved his condition somewhat so that on October 2nd I was able to operate. I examined him with the sigmoidoscope and found the rectum healthy, but many ulcers in the sigmoid. I removed the stump of the appendix and then, through the wound in the right iliac fossa, brought up a piece of ileum close to the cæcum and fixed it by a spigot through its mesentery. Six days later I opened it, establishing an artificial anus. Through this he discharged all his intestinal contents, and no fæcal matter passed over the ulcerated surface of the colon. He was irrigated through the lower end of the ileum without difficulty.

Sloughs had ceased to appear in the washes by Christmas, and his condition was so good that all washes were stopped on January 5th. As the artificial anus showed no signs of closing I excised the portion of ileum involved on February 5th, closing both ends; the lower was flush with the cæcum. I then carried out a lateral anastomosis of ileum to cæcum.

He is now (March 7th) quite well, he weighs 9 st. 5 lb., and his bowels are open daily and the stools normal.

These two cases illustrate the surgical treatment of ulcerative colitis—appendicostomy and irrigation; if this fails, or perhaps as a primary operation in advanced cases, ileostomy should be performed, the opening being made as close to the cæcum as possible to facilitate excision of the artificial anus and restoration of anatomical continuity when the

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ulcers have healed as the result of rest and irrigation.

*Chronic Ulcer of the First Part of the Duodenum Treated by Gastro-enterostomy.*

The patient, a man, æt. 28 years, illustrates the excellent results of the surgical treatment of chronic duodenal ulcer. While by no means every case of chronic gastric ulcer needs surgical treatment, I believe that chronic duodenal ulcer should always be treated by operation.

This man, who is an officer's servant, was sent to me from Aldershot two years ago, and has come up to-day to report himself. He gave a typical history extending over twelve months; he was never absolutely well, but after three weeks or a month's rest in bed he was well enough to resume work, only to find that he speedily relapsed.

I operated May 23rd, 1906. On opening the abdomen I found an indurated mass at the termination of the first part of the duodenum, adherent to the under-surface of the liver. I performed posterior "no-loop" gastro-enterostomy, making a two-inch anastomotic opening. He had no vomiting after the operation, and left hospital in three weeks and was back at his work five weeks after operation.

He has had no trouble of any kind since the operation, has put on a stone in weight, and eats all manner of food without even discomfort.

The scar is perfect and shows no sign of yielding. The abdomen was opened, as it always should be in operations upon the stomach and duodenum, behind the rectus muscle. The skin and anterior sheath of the right rectus were incised, the muscle pulled outwards and its posterior sheath and the peritoneum cut through. In closing the abdomen the posterior sheath and peritoneum were sutured with fine silk, the rectus stitched back into its place, and then the anterior sheath sutured with catgut. When the abdomen is opened and closed in this way no abdominal belt is necessary and all chance of a post-operative ventral hernia is avoided.

*Two Cases Illustrating Points in the Treatment of Appendicitis.*

This first patient, a man, æt. 40 years, was sent to hospital forty-eight hours after the onset of acute abdominal pain and vomiting. The pain came on suddenly while at work, was general and not at any time localised. He vomited almost immediately after the onset and continued to do so at frequent intervals up to admission.

When I saw him an hour after admission, it was obvious from his general appearance that he was suffering from an acute abdominal lesion. He was lying in the typical position, his tongue was beginning to become dry, his pulse was 110 and his temperature 99° F. The abdomen was rigid everywhere and deeply tender. A definite diagnosis was impossible, but owing to his age and the general involvement of the peritoneal cavity I inclined to the view that he had a perforated duodenal ulcer. On this diagnosis I opened the abdomen in the upper right portion and found it full of pus. Careful examination showed that the pus was welling up from below and the duodenum was normal; I accordingly rapidly removed a gangrenous appendix and drained through the right kidney-pouch, the mid-line and the right iliac fossa.

After the operation the head of the bed was raised until he was sufficiently recovered to be propped up, and *nothing* was given by the mouth. He had normal saline continuously by the rectum for forty-eight hours. His condition improved at once, his thirst disappeared, and his tongue became moist. Three weeks later when he appeared well, he was suddenly seized with acute abdominal pain of a colicky character, his temperature became subnormal, and he vomited large quantities on several occasions. I saw him within six hours of the onset, diagnosed acute intestinal obstruction and operated. On opening the abdomen a little clear fluid escaped and distended small intestine; on tracing this down it led to a coil of ileum strangulated by an omental band. The general peritoneal cavity, but for this adhesion, appeared perfectly healthy. This is now four weeks ago, and the patient is almost well.

The treatment and one of the after-results of peritonitis is illustrated by this patient.

Generalised suppurative peritonitis is by no means so fatal a disease as it was even three years ago; we are beginning to understand something of its treatment. Many cases—for instance, this man—now recover, that three years ago I should have considered hopeless. The lines of treatment are, rapid removal of the primary focus or closure of the perforation with as little manipula-

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tion as possible, followed by free drainage. No attempt at "cleansing" the peritoneal cavity by sponging or irrigation is justifiable in generalised peritonitis. Free drainage should then be provided. After the operation the head of the bed should be well raised and as soon as possible the patient himself propped up. Nothing should be given by the mouth, not even sips of water. This must be rigidly adhered to if the treatment is to be a success and continuous infusion of saline solution given by the rectum. This will have to be kept up for twenty-four hours at the least and in many cases for longer.

This next patient illustrates another of the complications produced by delay in surgical treatment. The last patient we saw nearly lost his life through twenty-four hours' delay; this patient has lost some weeks of valuable time, but has not been in any great danger. He is a man, æt. 42 years, and was sent to hospital on the tenth day of his first attack of appendicitis.

His pulse and temperature were normal, but he had a well-defined deeply tender swelling in the right iliac fossa, which was visible as well as palpable. It was a typical abscess, and I opened and drained it through a "muscle-splitting" incision over the most marked part of the swelling. Careful sponging and search with the help of a head lamp failed to reveal the position of the appendix. I try to remove the appendix in all cases of appendix abscess at the time of opening the abscess, but this is one of the cases in which prolonged search was not justifiable; the walls of the abscess were thick and it would have meant freely breaking down adhesions to find it; this is not in my opinion justifiable. This patient will return to hospital in about six weeks for removal of appendix. I consider that the treatment of no case of appendix abscess is complete until the appendix has been removed. I have on many occasions had to operate to drain a second or third appendix abscess. Time will be gained and the patient saved much inconvenience and risk if the rule is always made to remove the appendix at the time of opening an appendix abscess if this can be done justifiably, if not to remove it, at a second operation.

In both these patients operation was performed too late. In both more than three months will elapse before work can be resumed, whereas had it been possible to operate during the first twenty-four hours of the attack the length of treatment would probably have been under a month.

In all cases of acute appendicitis the appendix should be removed within the first twenty-four hours.

*Intestinal Obstruction with Ulceration of Cæcum due to Carcinoma of Sigmoid.*

This patient, who is now well and ready to leave hospital, has illustrated several interesting points in connection with intestinal obstruction. On his admission he illustrated the symptoms due to intestinal *obstruction* unassociated with strangulation.

Seven days before his admission he was taken ill with colicky abdominal pain, "as if his intestines were being tied into knots." Since that time he had passed no motion or flatus, but had been fairly comfortable, not strictly confined to bed, and had not vomited once. The abdomen was generally distended; this was most marked along the course of the large intestine, the cæcum in particular being very prominent; no visible peristalsis was noted. The abdomen in the right iliac region was rigid and deeply tender. The temperature was nearly 101° F. and his pulse 120.

An hour after admission I opened the abdomen in the mid-line below the umbilicus; blood-stained fluid came out and a much distended cæcum presented. On further examination I found a ring carcinoma of the sigmoid, quite operable. The cæcum was inflamed and there were greyish-green patches; at one of these perforation took place during my examination. I fixed the cæcum to the abdominal wall and drained it. He did well, and twenty-four days later I resected the sigmoid with the growth. Three weeks after this operation I restored the anatomical continuity of the intestine in the cæcal region. I made elliptical incisions around the artificial anus. After packing this with gauze, and cleaning the skin thoroughly while the patient was under the anæsthetic, I opened the peritoneal cavity and clamped the termination of the ileum and the cæcum immediately above the opening and then removed the cæcum and termination of the ileum. I closed the ends of the ascending colon and ileum and performed lateral anastomosis between the ileum and ascending colon.

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After being nearly three months in hospital the patient is now ready to leave.

The treatment of intestinal obstruction due to malignant stricture is well illustrated; in no acute case should any attempt at an "ideal" operation be made—the growth should never be removed and an immediate end-to-end anastomosis made. Drainage must always be carried out, and the anatomical continuity restored at a subsequent operation, but it will rarely be necessary to operate three times as I was forced to do in this case owing to the necessity of draining the inflamed cæcum—gut at a long distance from the seat of the disease.

*Radical Cure of Inguinal Hernia.*

This young man, æt. 21 years, has a right congenital hernia into the funicular process coming right down to the top of the testis. He wishes to have a radical cure performed, and as there are no contra-indications I propose to carry it out to-morrow, with the practical certainty that the operation will be a success and the cure "radical."

But in order to ensure success certain points must be remembered. Inguinal hernia is due in the large majority of cases to a congenital abnormality, the imperfect closure, to a greater or less extent, of the funicular process; a sac exists ready for the reception of the hernia. The essential of a rational operation consists, therefore, in the complete removal of this sac so as to leave no depression on the peritoneal surface opposite the internal ring. In order to avoid such complications as orchitis or the development of vaginal hydrocele later, the cord should be disturbed as little as possible. I consider operations of the Bassini type inadvisable: they possess all these faults; perfect obliteration of the sac is not always obtained, and the normal anatomical relations of the inguinal canal and the spermatic cord are unnecessarily disturbed. The simplest operation is here, as elsewhere, the best. I have for over four years performed a modification of Kocher's second operation. I open the inguinal canal by cutting through the external oblique aponeurosis and separate the sac from the cord without disturbing the latter. After it has been well freed up to the internal ring, opened and its contents reduced, it is pushed back into the peritoneal cavity and out through the internal oblique and transversalis muscles about an inch above and external to the internal abdominal ring. As it presents it is seen to be inside out with the peritoneal surface outermost. It is stitched to the muscles in this situation and cut off. If the hernia has been in existence for some time it may be necessary to stitch the lower edges of the internal oblique and transversalis muscles to the deep part of Poupart's ligament in front of the cord, but this is by no means essential in most cases.

This operation allows the patient to get up as soon as the wound is healed, and the patients are out of hospital on the seventeeth day, sometimes before. I have performed this operation over 150 times without any ill result.

*Cervical Ribs; Involvement of Superficial Nerves in Scar-tissue following their removal. Syringo-myelia in a Patient with Cervical Ribs.*

These illustrate certain points in connection with the surgery of cervical ribs. It is well known that, particularly in women, the presence of cervical ribs may give rise to wasting of the intrinsic muscles of the hand and changes in sensibility. These patients illustrate complications which may follow removal, and the possibility of mistaking the symptoms produced by syringomyelia for those due to the presence of a cervical rib.

The first patient, a woman, æt. 32 years, had cervical ribs removed from both sides of her neck at a provincial hospital. Since the operation she has had severe pain radiating from the centre of the incision on the left side over the shoulder, scapular region, and lower part of the face. This scar is, as you see, irregular, puckered, and adherent to the middle of the posterior border of the sterno-mastoid and the deep structures in this situation. She complains that the pain is always present and that it is increased by moving her head. You can see the attitude in which she stands, with the head so placed as to relax the sterno-mastoid of the affected side; this is the so-called protective torticollis. You also see the slightest movement of the head to the right pulls on the scar. The skin over the affected region is a little red as compared with that of the sound side, and on dragging the point of a pin lightly over the part from sound to affected portions it is easy to mark out a large area of tenderness corresponding to the full supply of the descending cervical nerves from three and four, with the transverse cervical and great auricular; the small occipital has escaped. There is no loss of any form of

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sensibility, light touch is well appreciated within the whole affected area, and she can easily distinguish warm from cool water. There is no doubt that these nerves were injured at the time of the operation, probably cut into, and now have become bound up in the mass of scar tissue which resulted from the suppuration which undoubtedly took place. I intend to-morrow to excise the scar in the skin, to turn forward the sterno-mastoid, and dissect out the superficial cervical nerves and deal with them according to the condition found; it may be necessary to resect portions of them. After dealing with the nerves I shall protect them from contracting adhesions to the skin and to the surrounding structures by wrapping them in Cargile membrane.

This is an example of irritative involvement of nerves in scar tissue as met with in civil practice; it demonstrates the widespread tenderness without marked loss of sensibility, and is an early stage of the more severe condition, causalgia, described fully by Weir Mitchell as the result of gunshot wounds of nerves.

This next patient, a girl, æt. 22 years, was sent to me on account of the wasting and weakness of the muscles of her right hand. I saw her in the Out-patient Department, and found that all the intrinsic muscles of the right hand were wasted, the change being most marked in the adductors of the thumb, which were also paralysed. The sex and age of the patient at once suggested the possibility of the symptoms being due to the presence of a cervical rib, and examination of the neck revealed their presence on both sides. This was well shown in the X-ray plate. I therefore took her into hospital for further investigation.

No person with nervous disease of any kind, no matter how apparent the diagnosis may appear to be, should be operated upon until the complete routine examination has been carried out; there is no rapid method of diagnosis for cases of this nature. After starting in the usual way with the obvious symptom of which the patient complained, the wasting of muscles, I tested sensibility; light touch, deep touch, and prick were everywhere normally appreciated, but on testing temperature sensation I found that over certain areas of the affected upper limb the patient was entirely unable to distinguish between ice and hot water. This at once negatived the possibility of the lesion being due to the pressure upon the trunk formed by the eighth cervical and first dorsal nerves. Heat and cold are never affected alone in lesions of peripheral nerves. This grouping pointed to a central lesion, which in this case is syringomyelia.

This is a disease you should always bear in mind in dealing with the symptoms supposed to be due to the presence of cervical ribs. The two are often associated, and I know of several instances in which cervical ribs have been removed for symptoms which proved later to be due to syringomyelia.

### *Chronic Gastric Ulcer.*

This woman, æt. 43 years, has, from her own account, been suffering from an "ulcerated stomach" for twenty years. She has all the classical signs of gastric ulcer, and I have no doubt that a chronic ulcer is present, probably on the lesser curvature, certainly not at the pylorus, for had this been the situation obstructive symptoms accompanied by dilation of the stomach would have been present long ago. This patient has been treated medically in an efficient manner, and her teeth have been attended to, and she gives the common history of improvement while at rest and dieted, relapse when again at work.

This is essentially a case in which operation should be performed. Medical treatment has been thorough, her mouth has been attended to, and the symptoms persist. No case of gastric ulcer should be treated surgically, unless, of course, for one of the disasters accompanying it, until all septic teeth have been removed and rest and diet thoroughly tried under medical supervision for a sufficient time. If a serious relapse occurs the sooner a surgeon is consulted the better.

To-morrow I intend to operate, and if definite physical signs are present in the stomach—and I have no doubt that they will be—perform posterior gastro-enterostomy without a loop. In cases in which an ulcer is present in any part of the stomach sufficiently large to be visible or palpable externally, gastro-enterostomy will certainly relieve the patient—in some cases bring about a cure. But you must remember and impress upon the patient that when the disease has lasted for many years, as in this case, it is unlikely that there will be absolute freedom from all stomach symptoms, the ulcer will heal, but the motility of the stomach will in all probability be interfered with somewhat by adhesions; the acute pain, vomiting, and hæmatemesis, however, will cease.

If, on opening the abdomen, no external sign of ulcer is present on carefully examining the whole of the stomach and duodenum, nothing in the nature of an operation should be performed on the stomach, and the cause of the symptoms must be looked for elsewhere before the abdomen is closed.

Gastro-enterostomy must never be performed in these cases on the off-chance that an ulcer is present, too small to be visible or palpable, or that it may relieve the gastralgia or atonic dyspepsia present; it will do no such thing, and may make the patient much worse. The most severe cases of regurgitant vomiting have arisen when this has been done. Gastro-enterostomy is the operation of choice when a chronic gastric ulcer is present.

*May 18th, 1908.*

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# THE CLINICAL JOURNAL,

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## A LECTURE ON LEUKÆMIA.*


By FREDERICK TAYLOR, M.D., F.R.C.P.,
Consulting Physician to Guy's Hospital; Physician to the Dreadnought Hospital, Greenwich.


GENTLEMEN,—Our subject to-day is leucocythæmia, or leukæmia. Leucocythæmia, as you probably know, is the name given to a morbid condition of the blood, consisting of a great increase of the white blood-corpuscles or leucocytes associated with disorder or disease of the blood-making organs: the spleen, the lymphatic glands, and the bone-marrow. But every case in which there is an increase of white corpuscles is not leucocythæmia. And we have to distinguish first of all between leucocythæmia and conditions known as leucocytosis. In health there are conditions of temporary increase of the white corpuscles, which may take place daily in consequence of the digestive processes. A temporary increase in the leucocytes also occurs in consequence of infectious disorders and other disturbances which do not last indefinitely. I may remind you of the proportions which the different kinds of leucocytes hold to the total number in health. The polymorphonuclear cells form from 50 to 60 per cent., the small lymphocytes 25 to 30 per cent., the large lymphocytes 5 per cent., and eosinophiles 1 to 2 per cent. In leucocytosis either the increase of leucocytes is one in which these proportions are maintained, or there is some excess of polymorphonuclear leucocytes, or in some exceptional conditions, as in certain stages of typhoid fever, there is a slight increase of lymphocytes. And while the number of leucocytes in health is 7000 to 8000 per c.mm. the number in leucocytosis is from 20,000 to 30,000,

* Delivered at the Medical Graduates' College and Polyclinic.

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or a little more. But what we have to do with in leucocythæmia is, in general, a much larger increase of the leucocytes, and one in which the normal proportions are very much disturbed, with the further fact that one finds in the blood corpuscles, which are not normally present, especially the cells called myelocytes or marrow-cells. Leucocythæmia, I say, is associated with enlargement or disease of the blood-making organs, the spleen, or the lymphatic glands, or the bone-marrow. The first cases were seen in connection with an enlarged spleen, and they were discovered independently by Hughes Bennett, of Edinburgh, and Virchow, in Germany. And after it was found that there were cases of leucocythæmia with enlarged spleen, other cases of leucocythæmia were observed in which the lymphatic glands were enlarged. Then it was discovered that in cases in which the spleen was enlarged the white corpuscles were somewhat different from what they were in cases of enlarged glands. It was Professor Virchow who made this distinction. He writes in his lectures on cellular pathology*: "I have therefore distinguished two forms of leukæmia, namely the usual splenic, and the lymphatic form, which nevertheless are often combined. The distinction rests not only upon this, that in one case the lymph glands, in the other the spleen, form the starting-point of the disease, but also upon the fact that the elements which occur in the blood do not entirely agree. While, for instance, in the splenic form, as a rule, the elements in the blood are relatively large, developed cells with single or multiple nuclei, which in many cases have a very great resemblance to splenic cells, it is seen that in the pronounced lymphatic forms the cells are small, the nuclei in proportion to the cells large and single, as a rule, sharply limited, with dark contours, and somewhat granular, and the cell membrane often lying so close that a space can be scarcely seen between them." He naturally regarded the elements in the blood as the direct result of the hyperplasia of the spleen and the lymph glands respectively, and he says in the same paragraph: "That is in brief the history of these processes." That was not indeed the whole history of the process, for not long afterwards it was discovered that the marrow of the bones was diseased as well as the spleen and lymphatic glands; and that suggested a third division. But it was soon found that even if they existed at all cases of purely bone-marrow leucocythæmia were extremely rare. And so it came to be the custom to distinguish two forms: (1) Splenomedullary cases, or cases in which the spleen and medulla were affected, and in which the white corpuscles were mostly large and unlike the so-called lymphatic corpuscles; and (2) purely lymphatic cases, in which the lymphatic glands were enlarged and the corpuscles were mainly a small variety of lymphocyte. However, there have undoubtedly been recorded cases in which the marrow of the bones was affected alone, without enlargement of the spleen or lymphatic glands, and leucocythæmia resulted. But the divisions which Virchow thought to be so simple and satisfactory do not now hold. The cases cannot be simply divided into three groups, namely, enlarged spleen with spleen corpuscles in the blood, enlarged glands with lymphatic or lymphoid cells in the blood, and diseased bone-marrow with marrow cells in the blood. In the cases in which the spleen is enlarged the corpuscles which predominate are myelocytes—corpuscles derived from the marrow of the bones and not from the spleen. In many cases in which lymphoid cells, or lymphocytes, are abundant in the blood there is no enlargement of the lymphatic glands. So that it does not appear to be at all necessary for us to believe that lymphocytes are derived solely from the lymphatic glands. And thirdly, and perhaps most important of all, a much larger acquaintance with these cases shows that in every kind of leucocythæmia, whether the lymphatic glands are enlarged or the spleen is enlarged, or both together, or neither, the bone-marrow is always diseased. It therefore becomes clear that the bone-marrow can provide every kind of corpuscle in excess, and that it is chiefly responsible for the disease of the blood known as leucocythæmia. These last facts have rendered it almost essential that we should consider the classification of the different cases of leucocythæmia from the point of view of the blood, and not from that of the organs diseased. And clinically, as an outcome of that, it results that no satisfactory diagnosis of leucocythæmia can be made without an examination of the blood itself—that is to say, no recognition of the fact that a patient has an enlarged spleen is to lead you to make a hasty diagnosis of leucocythæmia. Nor can you say

* 'Die Cellularpathologie,' Berlin, 3rd edition, p. 161.

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that because a person has not an enlarged spleen or enlarged glands therefore he has not leucocythæmia. The ultimate judgment and diagnosis in these cases must depend on an examination of the blood, and nothing can be done in this direction without it. For purposes of nomenclature, as I have said, the present view is that we must divide the different cases according to the condition of the blood. Therefore it is desirable to drop the term "spleno-medullary leucocythæmia," because it is named after the organs involved. It has been called *myelocythæmia*, because the predominant leucocytes are myelocytes, and it has been called *myelæmia*, which is a shorter term. On the other hand, lymphatic leucocythæmia has been called *lymphocythæmia* or *lymphæmia*. But the last word has the disadvantage that it may suggest an association with the lymphatic glands, whereas it is desirable to confine the names to an association with the corpuscles. Probably the best terms which have been suggested for the two groups, for the present at least, are *myelocytic leukæmia*, in which myelocytes are the predominant leucocyte, and *lymphocytic leukæmia*, in which lymphocytes are the predominant leucocyte. One has to recognise that there are mixed cases—cases which do not accord accurately with either of these two types. But it is convenient to deal first with those conditions which are most frequent and most typical, and to recognise that there are other kinds which are met with less frequently.

I may remind you of the features of the different leucocytes:

The small lymphocyte is a cell having a relatively large, generally darkly-stained nucleus and a comparatively small amount of protoplasm. In another form of leucocyte the whole cell is larger and the proportion of protoplasm greater. In neither of these does the protoplasm contain granules; they are sometimes called non-granular corpuscles. Then there are cells whose protoplasm is granular, granular corpuscles, of which the most familiar is the polymorphonuclear leucocyte, the granules being described as neutrophile. Then there are eosinophiles in which there are large granules, staining with acid dyes, and especially with eosin; and here, on the other hand, I show you a diagram of a so-called mast-cell, in which the granules are large and stain with basic dyes, a cell which is occasionally present, but not generally in large numbers. Next I have to mention the myelocyte, which is not present in normal blood, but is present in the marrow of bones. This is a large cell not unlike a large lymphocyte, with a large oval or horse-shoe-shaped nucleus within it, and with some neutrophile granules; myelocytes also occur in which eosinophile granules are present, and some with basic granules like the polymorphonuclear mast cell. The most common form of leucocythæmia, myelocytic or splenomedullary leukæmia, that which was first described, is that in which the leucocytes themselves may reach 200,000, 600,000, or even as many as 900,000 to the millimetre, and the proportion of the corpuscles is often from 40 to 50 per cent. polymorphonuclear leucocytes, from 30 to 40 per cent. myelocytes, with only from 4 to 5 per cent. lymphocytes, and from 2 to 4 per cent. eosinophiles, which you will see are somewhat increased. And such proportions are constantly observed. Under one of these microscopes you will see a very good specimen of that kind of blood, representing a large number of myelocytes, a good number of polymorphonuclear leucocytes, and a diminished number of lymphocytes.

As to the ætiology of this condition practically very little is known. The conditions occur in persons of both sexes, and especially at the period between later childhood and puberty, through to middle age, and they may even be found in persons of advanced age. One striking feature about these cases is that the onset is often quite insidious. It is a common experience now to find a patient gets out of health and yet not sufficiently so to give up work or even to consult a medical man. He finds his abdomen getting very distinctly larger, but other parts of his body do not increase proportionately. When at length he does consult a medical man it is found that his spleen is already enormously enlarged, and when the blood is examined the leucocythæmia is found to be already very pronounced. The blood is somewhat paler than normal, because the red cells are in very diminished numbers, such as 3,500,000 instead of about 5,000,000, or even lower, while the leucocytes number from 200,000 to 250,000 or even more.

The patient is weak, short of breath, and somewhat pale. But it is important to remember that these patients do not necessarily present a pallor which is at all proportionate to the

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disease of the blood from which they suffer. The mucous membranes are not necessarily white or strikingly anæmic. The lips of these patients are quite often of full colour, and in that respect they may be very deceptive, so that it may be regarded as quite impossible that they are the subjects of such a disease as they have. There may be, and there is frequently in course of time, a little swelling of the feet. There is some emaciation of the limbs and face, while the abdomen is enlarged, and there is frequently some pyrexia. This pyrexia is not generally very high, but is variable and may increase for a time and diminish again, and again increase. In those cases the enlargement of the spleen is generally characteristic. It may present a comparatively sharp edge towards the median line, which it generally fails to reach above the umbilicus, but across which it may pass below that point. In this edge is a notch, or perhaps two notches. The organ is dull all over, and smooth, coming as it were down from the costal margin, and pushing the hollow viscera to the right of it, but never overlaid by the colon. It is always continuous with the rib surface, and therefore unmistakable in that way. The liver is frequently somewhat enlarged, transcending the costal margin by two or three inches. Those cases are variable in their course. Sometimes they are relatively rapid, and the patients sink in the course of five or six months from the first appearance of the complaint. In other cases they may go on for two or three or four years. It has been commonly said that the average duration of life of patients with this disease is about two years. But averages in this respect are not very valuable; what you want to know are the extremes. The disease rarely lasts from the time of its first recognition more than four years. Possibly in consequence of treatment which has been recently carried out the duration of these cases may be increased.

In their subsequent course, these cases present many resemblances to Bright's disease. The patients may have hæmorrhages in various parts, dropsy, effusions into the serous cavities, serous inflammations such as pleurisy and pericarditis, dilated heart, and a form of retinitis which presents a close resemblance to that of Bright's disease, and which is especially characterised by considerable tortuosity of the vessels of the retinal area, with moderate swelling of the optic disc. Probably it is due to lymphatic infiltration of the optic nerve and compression of the vein, so that its return of blood is impeded. Death takes place eventually from increasing prostration, failure on the part of the heart and the subsequent occurrence of these inflammatory and dropsical conditions which I have just mentioned. The spleen is generally found, on section, to present a number of fibrous trabeculæ, the Malpighian corpuscles are obscure, and the splenic pulp is literally stuffed with numerous leucocytes of all types, myelocytes and polymorphonuclear cells, or lymphocytes, but especially those which are represented as most predominant in the blood. The bone-marrow is altered in this condition, the cells are present in great number, and there are numerous myelocytes. The liver is very generally infiltrated or impregnated with leucocytes of the same kind as are present in other parts.

The diagnosis of this condition ought to be extremely easy. I have already referred to the fact that an early diagnosis is not common because apparently the conditions make so little impression on the patient. If the spleen is enlarged of course it must be eventually recognised, and I pointed out two features which are characteristic of it. I hardly like to refer to the fact that mistakes can be made in the recognition of a spleen such as I have depicted, and yet it has been done on more than one occasion in the past. In a female patient the spleen has been mistaken for an ovary. Such a case has been sent to the hospital for the treatment of ovarian tumour. I can imagine that the mistake might have been made partly in consequence of the fact to which I have referred, that the patient with leucocythæmia and enlarged spleen does not always show that pallor which perhaps the observer thinks ought to be present with such a disease of the blood. And so the conception of ovarian tumour in the female patient was allowed to be entertained. Of course there are other enlargements of the spleen with which this can be confounded, and here again I would impress upon you the necessity of examining the blood before any diagnosis is made. One of these is known as splenic anæmia, and in this disease the spleen may be nearly as large as that found in myelocytic leukæmia, but the blood presents no excess of leucocytes. There is, indeed,

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a diminution in the red corpuscles, but the leucocytes are somewhat less than normal, being as low as 4000 or 3000 instead of 10,000. There are cases also of enlarged spleen in which the conditions neither of splenic anæmia nor of leucocythæmia are present; in which, indeed, the blood is apparently perfectly normal. These cases are still obscure and much has to be learned about them; but they are certainly not so quickly fatal as either of those above mentioned. The diagnosis, then, must depend on the examination of the blood, and not only upon that of the viscera.

The next kind of leucocythæmia which I shall refer to is the so-called lymphatic leukæmia. Here the blood picture is very different. The lymphocytes are increased. There may be the same excess in the number of leucocytes as in the former cases, namely from 200,000 to even 800,000. In the most typical of these cases the lymphocytes are increased so much as to be 90 to 95 per cent. of the whole. The remaining corpuscles are mainly polymorphonuclear corpuscles, and there may be only a few myelocytes, perhaps not more than 1 per cent. to 2 per cent. This is a very striking condition, and one of these slides shows it under the microscope. Nearly the whole field appears to be filled with leucocytes of about the same size, obviously showing the characters of the lymphocyte and justifying the name "lymphocytic leukæmia." Of that class of case there seems to be more variety than there is amongst the myelocytic. First of all, there is a group of cases which have been called acute leukæmia, or acute leucocythæmia, in which the patients have generally had, as the first symptom, some ulceration of the mouth, such as stomatitis, or gingivitis. Then they have become pale and weak; the temperature has been raised, and then there has appeared some enlargement of the glands, or perhaps enlargement of the spleen. These patients have quite early suffered from hæmorrhages, so that observers have often had in their minds the existence of purpura, or scorbutus or other obscure blood diseases. These again are cases in which an examination of the blood is essential in order to arrive at a correct diagnosis. In these cases it is generally the large lymphocytes which have been increased in number, the small lymphocytes being comparatively few. They are very fatal: patients with this condition have died at as short a period as two weeks from the first urgent symptoms, and many have died in three or four weeks

There is another group of cases which is not very different from them, but in which the duration is not quite so short. This form of the disease does not necessarily begin with these conditions of stomatitis and gingivitis which I have mentioned; but one or more of the glandular organs become enlarged, such as the lymphatic glands, the spleen, liver, and thymus; and the racemose glands, namely, the salivary, mammary, and lachrymal glands; and though the blood contains excess of lymphocytes the lymph-glands are not necessarily enlarged either first or most. The specimen under the microscope is that from a girl who had an illness which was fatal in from three to four months. I saw her about the middle of the third month, and then she had an enlarged spleen, an enlarged liver, enlarged mammæ—enlarged, as I had good reason for believing, on account of the disorder and not from mere idiosyncrasy—and enlarged parotid glands. But there was no marked enlargement of lymphatic glands so far as they could be reached, though it is possible some of the internal glands may have been affected. There were also hæmorrhages in the retinæ. The smear of blood from her case shows an enormous number of lymphocytes, amounting to 99·5 per cent. of the total leucocytes. Of these lymphocytes 95·8 per cent. were small, and the remainder, 3·7 per cent., were of the large type. She died a few weeks later, but no post-mortem was obtained.

Thirdly, there are cases of chronic lymphocytic leukæmia in which the course of the disease is much more slow and prolonged. Here also the lymphocytes are chiefly of the small type, and with much more certainty will you find the lymphatic glands prominently enlarged and diseased. No doubt those are cases which correspond to those first described as lymphatic leucocythæmia. I do not know that they present any particular features which are different, so far as the general condition of the patient is concerned, from the leucocythæmia of other types. The patients become more or less anæmic in the course of time, they have the general condition of failure of all the organs, which is present in all cases of pronounced splenomedullary or myelocytic leucocythæmia, and the prognosis is very unfavourable. An interesting point with regard to this has to be considered, and

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that is: What is the date at which, in these chronic lymphatic leukæmias, the leucocytes are seen in the blood? In the disease known as lymphadenoma, or Hodgkin's disease, the first change is an enlargement of the lymphatic glands. This generally begins in the cervical glands, and subsequently the axillary or the inguinal are affected; at the same time or later the glands in the interior of the body, such as the mediastinal and the retro-peritoneal glands, become involved. And the spleen is not infrequently enlarged, and often it presents large masses of lymphadenomatous tissue in its interior. In some cases also deposits of lymphadenomatous tissue occur in different parts of the body, far from the spleen or lymphatic glands, but developing in the lymphoid tissue of other parts. Now, in typical Hodgkin's disease there is no alteration in the condition of the blood other than mere anæmia. There is a diminution in the red corpuscles, but the leucocytes are not affected in number or proportion. But it has to be admitted that some of these cases, in their later stages, develop a lymphatic leucocythæmia. As some writers would express it, the leucocytes accumulated in the organs eventually overflow into the blood, and a case which has been originally lymphadenoma or Hodgkin's disease becomes one of lymphocytic leucocythæmia. Nevertheless, though such cases happen sometimes the majority of cases of lymphadenoma or Hodgkin's disease are fatal without ever becoming cases of leucocythæmia. So for the present we must recognise that the two conditions are distinct and separate. But in considering the group of lymphocytic leucocythæmias we must recognise one division of this group to consist of cases arising out of previous Hodgkin's disease.

The prognosis of cases of lymphatic leucocythæmia is, of course, very bad also, and the acute cases are at present hopeless. The longer they last, however, the better chance there is of some improvement happening.

With regard to treatment, I think we must admit that the outlook with regard to leucocythæmia is somewhat better at the present day than it was some time ago, when one had the impression that cases never recovered. Undoubtedly improvement has been effected by the administration of arsenic, in the form of the liquor, in increasing doses, and as long as the patients can take it. The longer they are able to take it without being upset by it, the better chance there is of some improvement taking place in their condition, as regards both the number of leucocytes and the enlargement of the spleen. Numerous other drugs and other treatments have been tried at different times, among which may be mentioned inhalations of oxygen, after the use of which in a case under my care there was a considerable diminution in the number of corpuscles and in the size of the spleen. That patient was, however, taking moderate doses of arsenic at the same time. I was never quite so successful with any other case. But in the present day the treatment which has been very largely employed has been that by means of X-rays. They have been directed upon the spleen and upon certain of the bones, especially the lower or epiphysial ends of the femur, as presenting a large surface and giving the best chance for the rays to influence the substance of the marrow. And a good deal of evidence has been forthcoming to the effect that patients have benefited very considerably by that means. Here I may confess that I myself have not been entirely successful. I had one case some time ago in which it is true the patient did not go backwards under the treatment, but he remained stationary, and there was no appreciable alteration in the number of the leucocytes, corpuscles, or in the size of the spleen; but he did not get progressively worse. So it may be that the treatment operated favourably though slowly. In the case of a lady whom I saw last summer, there was very pronounced leucocythæmia of the myelocytic type; and the spleen was very much enlarged. She was submitted to treatment by X-rays, but they upset her very much indeed, and she did not continue the treatment for a very long time. I had, however, this information about her, that the spleen appeared to diminish in size considerably, and to that extent she was improved. But the case is not cured yet. You will find, nevertheless, a good many cases recorded in the last two or three years, both in England and abroad, in which there has been improvement in consequence of this treatment. I think, therefore, it is one which we should all feel it our duty to employ. At the same time, a good deal of care must be exercised. It appears, especially in one or two cases, that the white corpuscles have been reduced almost to the normal in the course of treatment, but when the treatment has been

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continued after that stage was reached the patients have been rendered worse. Apparently the influence of the X-rays upon the life or the formation of the leucocytes having been carried beyond the point at which a normal result was obtained, prejudicial effects have been produced.

With regard to the theories concerning leucocythæmia there is much still to be learned. What seems to be clear is that, as I have implied, we are not to regard each form of leucocythæmia as the immediate result of a disorder of any one particular blood-forming organ, such as that, if the spleen goes wrong it is one kind, that if the lymphatic glands go wrong it is another, and if the marrow of the bones is disordered it is a third kind. How far, in some of the varieties, the spleen and the lymphatic glands contribute to the excess of corpuscles seems to me a very open question. The general impression appears to be that the original leucocyte, the parent cell in the bone-marrow, is a large cell, and that it develops into myelocytes or polymorphonuclear leucocytes—the granular cells—on the one hand, and into cells of the non-granular type on the other; that in the granular type the myelocyte precedes the polymorphonuclear cell, and in the non-granular type the large lymphocyte precedes the small one. Further, it may be supposed that the disease of the marrow in different cases produces these leucocytes at different rates and in varying quantities, and thus causes the different forms, though it is not quite clear exactly why these varieties should be. Experiments have been performed which show that the exhibition of various toxines influences the marrow in different ways, some affecting the production of leucocytes in one way, and others in other ways. But whether, in the natural occurrence of the disease, such toxines are introduced into the body, and what their nature is, remains doubtful. With regard to the enlargement of the organs, it has long been regarded as probable that the spleen and lymphatic glands were not large simply because they were hypertrophied and morbidly active, but rather that they were stuffed with the leucocytes which were produced in excess by the marrow. And this view is supported by the facts that the bone-marrow may be diseased in every variety of leukæmia, and that in some cases of lymphocytic leucocythæmia the lymph-glands are not primarily enlarged.

*May 25th*, 1908.

# SOME INTERESTING CASES OF CRANIAL SURGERY.

A Lecture delivered at the Polyclinic, London.

By HENRY CURTIS, B.S., F.R.C.S.,

Assistant Surgeon to the Metropolitan Hospital, Kingsland Road, N.E., and to the Branch Seamen's Hospital, Royal Albert Docks, E., and Senior Clinical Assistant in the Ear and Throat Departments of the London Hospital, E.

(*Concluded from p.* 91.)

CASES 5 and 6 illustrate the meningitis due to the pneumococcus, to which variety alone we shall have time to allude. It is almost always secondary to disease elsewhere, complicating pneumonia and endocarditis, and results also from infection from the nose, throat and ear. It occurs at all ages, runs its course in from a few days to a week or ten days, and is invariably, or almost invariably, fatal.

CASE 5.—*A case of pneumococcic meningitis supervening upon pneumonia.*—This case, which was under my care when in South Africa, perhaps should not be included under the title of cranial surgery. It serves, however, to illustrate a condition with which every surgeon should become familiar, since the diagnosis may, as in Case 6, have to be made from other varieties of cranial disease, even if the time has not yet arrived for classing all forms of meningitis amongst cases amenable to active treatment. The temperature charts on the first and second admissions to hospital have been combined for comparison of the two conditions.

A bright, alert Matabele boy, æt. about 11 to 12 years, came under observation on the morning of July 25th, 1905, with pain referred to the sternum, later to the left side of the chest. T. 103·2° F., P. 100, R. 24. The tongue was thickly coated with light brown fur. There was no abdominal pain or gurgling, and the stools were normal on the night of admission, when the temperature had fallen to 98·6° F.

The chest showed no dulness, and the only sign to be detected was some friction in the axilla and left back. The case was treated as for pleurisy and pneumonia, but the diagnosis of enteric fever, then prevalent, was also entertained.

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The next morning, T. 101·1° F., P. 108, R. 28. In the evening the temperature was 103·6° F., and there were now signs in the right back similar to those noted on the left side on admission.

On the following morning, July 27th, the temperature was normal; the patient recovered remarkably rapidly; he insisted on working in the ward, and being anxious to return to his master's employment he left the Hospital on July 30th, against advice, but apparently quite well, though not of robust physique.

Eight days later, on August 7th, the boy was brought back, complaining of frontal headache, with T. 102° F., P. 96, R. 40.

result could not be investigated, as the boy escaped from the ward.

By five p.m., T. 104° F., P. 106, R. 38, in spite of antipyretics, including quinine, gr. x, 4 *tis horis*; and there was such a degree of collapse that ammonia, ether, and strychnine, with brandy, were prescribed. An hour later the boy became restless, but during the night slept heavily.

The next morning, T. 103° F., P. 100, R. 30. The boy rambled, and could not be induced to protrude the tongue. The lips were dry, and there was internal strabismus of the left eye. Bowels not open.

*Chest.*—There was no evidence of active

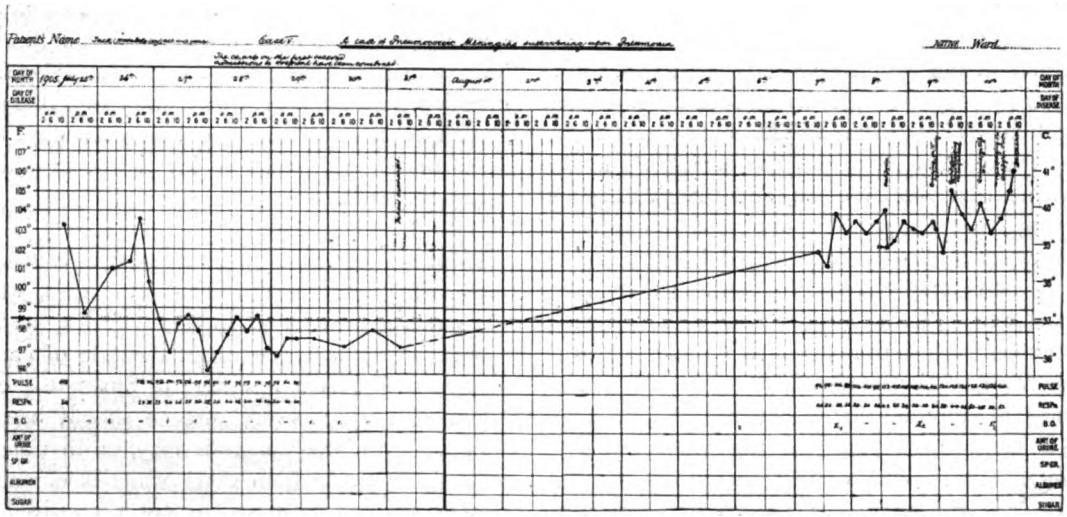

FIG. 8.—Combined temperature chart, Case 5. Pneumococcic meningitis following pneumonia.

Lips covered with sordes; tongue dry, cracked and covered with brown fur.

There was no tenderness anywhere in the abdomen, nor gurgling in the right iliac fossa.

The spleen was enlarged, but in natives this is frequently so—almost invariably so, one might say—owing to malaria.

There were no physical signs in the chest, except those of thickened pleura at the left base and side.

The diagnosis then appeared to be influenza, or possibly enteric fever, suspected when the boy was admitted previously, but then negatived by observation of the stools and the clinical course.

As there was some constipation, an enema was given on the morning of re-admission, but the mischief in the lungs, back or front. The heart appeared to be much dilated, and the sounds were very loud. There was no bruit.

August 9th: The patient slept heavily last night, but to-day there is muttering delirium. There is proptosis of the left eye. The morning temperature was 103° F. A partly-formed dark brown stool, not typhoidal, though there was some blood visible, resulted from an enema to-day. The boy's aspect appeared typical of the "typhoidal state," and the diagnosis of basal meningitis was now first entertained. At 5 p.m., T. 105·2° F., P. 128, R. 44 (in spite of a hypodermic injection of quinine, gr. v, at 10 a.m.).

At 10 p.m., T. 104° F., P. 130, R. 40, after tepid sponging, ice-bag to nape of neck, phenacetin and caffeine.

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On the following morning, August 10th, at 6 a.m., T. 104·6° F., P. 128, R. 44, falling by 10 a.m., as the result of hypodermic quinine, gr. vii, to 103° F., P. 130, R. 40.

*Examination of the chest this morning.*—Tubular breathing at the left base behind, and in the axilla. There were no crepitations, except bronchitic râles at the root of the left lung (onset of pneumonia).

Right lung normal. Pulse rapid but strong.

At 1.30 p.m. the temperature was 103·8° F., and it continued to rise in spite of tepid sponging and phenacetin.

At 3.30 p.m. a dark brown, non-typhoidal stool resulted from an enema. Temperature 105·2° F.

At about 5 p.m. there was a marked change for the worse, the boy becoming moribund. Pulse very weak, and difficulty in disposing of the mucus expectorated.

Proptosis of both eyes—right eye rather more affected than the left. Internal strabisms. Occasional groans.

Death at 6.15 p.m.

*Autopsy.—Cranium*: On removing the calvarium, extensive purulent meningitis was evident all over the vertex and base; there was a large mass of lymph at the base, extending to the foramen magnum, but not into the spinal canal.

Films and cultures from the lymph showed pure culture of typical pneumococci.

*Chest*: Recent left-sided pneumonia was seen, evidently a recrudescence of old mischief.

*Abdomen*: Examination of the intestines negatived the presence, at any time, of enteric fever.

CASE 6.—*A case of acute purulent pneumococcic meningitis, with lateral sinus pyæmia, from infection through the tonsils; absence of rigors throughout; operation; death.*—The patient, a boy, æt. 6 years, was admitted on May 15th, 1907, to the Metropolitan Hospital with the following history, for the greater part of which I am indebted to the courtesy of my colleague, Dr. Langdon Brown, as also for valuable suggestions and comments.

On April 23rd he began to suffer from a sore throat. He was seen by Dr. L. O. Young, who found the tonsils much swollen, but without exudation. The temperature was 103° F. and he was slightly delirious. Three days later the temperature had fallen to normal, and the child seemed almost well. He then began to suffer from headache, referred first to the forehead, then to the right parietal region, with some photophobia, and the temperature rose again.

On May 8th he was seen by Dr. Langdon Brown, in consultation with Dr. Young. There was no optic neuritis, no aural discharge, nor mastoid tenderness, no paralysis or alteration of the reflexes. Nothing abnormal was found in the chest or abdomen. There had been no vomiting throughout. Widal's reaction was negative. The headache improved for a time after this, and the temperature fell. A week later there was a recrudescence of temperature, headache, and tonsillitis, so he was admitted to the hospital under Dr. Langdon Brown.

There was nothing in the past history beyond "inflammation of the lungs" when ten months old, and measles and varicella when two years old.

May 15th, 1907: On admission he was found to have wasted rapidly. He lay curled up in bed on his left side, but there was no retraction of the head. There was no squint, and no ocular palsy. The pupils were equal, of medium size, and reacted to light and accommodation. Optic neuritis was now present in both eyes, there being about 3 D. of swelling. No paralysis of any cranial nerve could be found. Both tympanic membranes were natural, but the boy's hearing was impaired on the right side. There was still no mastoid tenderness or œdema. The tongue was furred and the tonsils were swollen, especially the right. There were some tender glands under the jaw on that side, and tenderness along the right sterno-mastoid. Chest and abdomen were natural. Kernig's sign was present. His pulse was 120, and his temperature 103° F. A blood-count on May 17th showed 22,000 leucocytes. Throughout there had been no rigors or vomiting. He was conscious and intelligent, and took food well, but there was evident intra-cranial mischief, and infection through the throat seemed the probable cause. A surgical opinion was therefore sought, and on Saturday evening, May 18th, when the boy first came under my notice, the general condition of the patient was seen to be as just described.

*May 18th.*—The patient complained of pain around (behind and below) the angle of the right inferior maxilla, where there was a very tender

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mass of enlarged glands, and the adjacent side of the skull, immediately below the tip of the mastoid.

On examination, the right membrana tympani was confirmed to be intact and normal. Nor was there any redness, œdema, or tenderness of the skin over the mastoid on either side.

Associated with the pain around the angle of the jaw there was marked tenderness along the anterior border of the right sterno-mastoid muscle, extending below almost to the right sterno-clavicular junction, and increasing in severity on proceeding upwards to the mass of glands below and behind the angle of the jaw, where the tenderness was extremely acute.

Some soreness of the throat was complained of, and the tonsils were found to be a little enlarged, and, with the fauces generally, decidedly congested, though there was no follicular tonsillitis, nor any membrane visible.

*As to the diagnosis.* — My colleague inclined towards the view of intra-cranial suppuration secondary to mastoiditis, and suggested exploration of the mastoid, and should nothing be found, examination of the lateral sinus; and the exquisite tenderness along the anterior border of the sterno-mastoid, with the other symptoms and signs, certainly favoured the idea of right lateral sinus thrombosis, extending downwards along the right jugular vein.

However, the normal state of the right membrana tympani, and the absence of redness, œdema, and tenderness over both mastoids, tended to discount, somewhat, the possibility of mastoiditis as the source of the intra-cranial symptoms, evidenced by the pain and double optic neuritis. The fact that well-established cases of suppurative inflammation of one or both mastoids have been observed without known otorrhœa, past or present, was, nevertheless, clearly borne in mind, especially in view of the tenderness along the whole of the sterno-mastoid muscle.

This tenderness in the region of the enlarged glands at the angle of the jaw, however, was so intense compared with that observed elsewhere along the sterno-mastoid that, in the absence of middle-ear disease and mastoiditis, it was attributed to irritation of branches of the superficial cervical plexus, set up by the lymphadenitis.

This condition of the glands was seen to a less extent also on the left side of the neck.

My view was that the symptoms pointed rather to tuberculous meningitis, the more acute symptoms being caused by the lymphadenitis.

It was therefore decided to postpone exploration of the mastoid, etc., meanwhile performing an exploratory lumbar puncture. This was undertaken the same night (May 18th). Clear watery fluid in considerable quantity, and under some pressure, was collected, and culture-tubes inoculated, with negative results. The pathologist, Dr. E. H. Shaw, also noted that the cerebro-spinal fluid evacuated contained leucocytes, 40 per c.mm., nearly all lymphocytes, very few polymorpho-nuclears, and no tubercle bacilli. This certainly somewhat favoured the diagnosis of tuberculous mischief, but, as we shall see, too much weight must not be attached to the negative results of lumbar puncture.

On the day following the lumbar puncture (Sunday, May 19th), and probably as the result of some relief of tension, the boy seemed decidedly better, and the temperature had fallen to normal.

It soon rose again, however (103° to 104° F.), assuming a hectic type for several days. Though the throat improved, and the glands were subsiding, the tenderness along the sterno-mastoid continued, and a blood-count on May 21st showed the leucocytes to have increased to 24,000. The boy became more irritable, occasionally crying out, and starting in his sleep.

It was felt, therefore, that there were points against the diagnosis of tuberculous meningitis, and the need for exploration had become urgent.

May 21st, 1907: *Operation* 1 (by Mr. Curtis). —The right mastoid antrum was opened in the evening, and found to be quite healthy. The mastoid emissary vein bled freely.

The lateral sinus was then exposed, and found to be partially thrombosed, the clot being discoloured, and of a brownish-black tint. Immediately above the exposed sinus the dura, observed to be bulging, was incised, and a large collection of thick, greenish-yellow, odourless pus shot out in a jet several inches high, and continued to escape for some seconds. The bone was removed freely with a view to following up the pus, and securing free drainage.* Smart hæmorrhage

* At the autopsy it became clear that removal of the entire right parietal and half of the occipital bone would hardly have been sufficient, even were that feasible.

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from the lateral sinus behind the clot exposed in the trephine hole showed the sinus to be patent up to that spot. The right jugular vein was then divided between two ligatures in the neck.

The skin wound in the neck was sutured, and healed by primary union. The incision through the dura mater was drained with gauze, a plug of gauze being also inserted into the opened up mastoid antrum to prevent infection. The pus removed from beneath the dura was found to be teeming with typical capsulated pneumococci.

One may here with advantage pause to emphasise the remarkable facts (1) that so large a collection of pus lying beneath the dura, and evidently under great pressure, should have caused neither paralysis, nor vomiting, nor did the pulse give the least evidence of compression; and (2) the absence of rigors, in spite of the marked sigmoid thrombosis.

The next day (Wednesday, May 22nd) the general condition seemed improved. There was no headache or vomiting; food was taken well. At 6 a.m. T. 100° F., P. 128, R. 28; at 10 p.m. T. 98·5° F., P. 128, R. 24. The wound was dressed, the plug of gauze being removed, and the mastoid antrum found to be quite dry and sweet. On removing the plug from the lateral sinus sharp hæmorrhage ensued, checked by a fresh plug.

There was no swelling in the neck.

Up to May 24th the boy remained bright and intelligent, eating and sleeping well; there was no headache, or vomiting, or rigor, the average temperature being about 99° F.

By midnight, May 25th, the temperature had risen to 102–103° F., and drowsiness, present earlier in the day, had become marked. There was head retraction, but no headache, rigor, or vomiting.

May 25th: *Operation* 2.—At midnight, in my absence, my colleague, Mr. Gordon Watson, explored thoroughly in various directions, but with negative results.

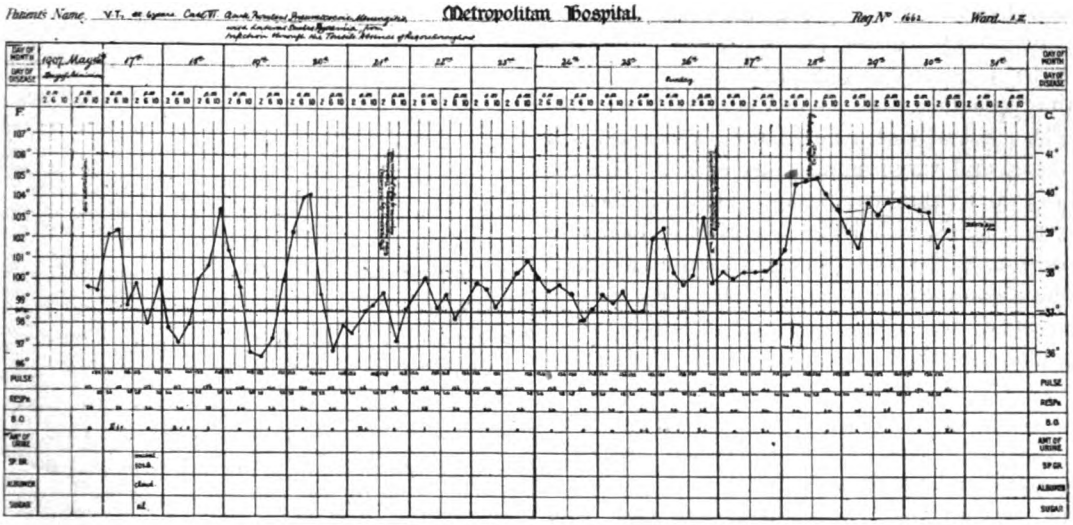


FIG. 9.—Temperature chart, Case 6. Pneumococcic meningitis, with lateral sinus pyæmia. Infection through the tonsils. Absence of rigors throughout.

On the following day, and for the first time (and possibly only due to the anæsthetic), the patient vomited once and was drowsy, but could be roused.

The meningitic cry, head retraction, and fever became more pronounced from this date, May 27th to 30th, when the pulse became weak, rapid and irregular. At midnight the respiration-rate increased to 52 per minute, but nothing abnormal could be detected in the chest. Death occurred on May 31st, about 8.30 a.m., the pulse being uncountable, and rigors having been absent throughout.

Beyond the evidence of lateral sinus thrombosis, with definite meningitis, and much pus beneath the

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dura, until the autopsy an exact diagnosis could not be arrived at, and this will not be a matter of reproach, perhaps, when the conflicting ante-mortem observations are considered in relation with the somewhat unusual post-mortem findings.

*Autopsy.*—By Dr. E. H. Shaw, Pathologist to the Metropolitan Hospital, etc. May 31st, 1907. (Examination of brain only permitted.)

Body very wasted.

Over rather more than the posterior half of the right side of the brain, from the right occipital pole to just in front of the motor area, the condition was one of acute suppurative meningitis, there being numerous pockets of pus, some of about the size of the terminal phalanx of the thumb, and larger, localised by very firm adhesions between the dura and pia mater, and separated from each other by these and by areas not obviously affected with suppuration.

The cerebral cortex under these abscesses was flattened and very soft, and tore easily. The left half of the cerebral cortex showed slight but extensive meningitis, with lymph exudations, evidently representing the earlier phases of the advanced stages seen on the right side. There was a large amount of free watery pus and lymph over the whole base of the brain, and the choroid plexuses were thickened and matted with pus.

A clot, breaking down in places, partially occupied the right lateral sinus, and pus was expressed from within and around the origin of the right jugular vein, which had, however, been divided in the neck below between two ligatures ten days earlier.

The only other lesions evident in this case (the autopsy having been limited, as stated) were the acutely inflamed tonsils and the mass of enlarged glands at the angle of the right lower jaw, glands on the other side of the neck also being a little enlarged.

*Bacteriological examination* (by Dr. Shaw).—Films and cultures from pus from beneath the dura, obtained at Operation 1, showed pure pneumococci, cultures in broth, milk, saccharose, and raffinose (creating an acid reaction, as indicated by the altered colour of the litmus) giving a positive result, that with salicin and mannite being negative.

Films and cultures obtained from the brain at the autopsy also teemed with pneumococci.

## Comments.

*As to the diagnosis.*—The tenderness along the right sterno-mastoid muscle suggested lateral sinus pyæmia, but this was rendered somewhat doubtful owing to the presence of a large mass of exquisitely tender glands at the angle of the jaw, having as their most obvious source of infection the enlarged and acutely congested tonsils. The tenderness along the sterno-mastoid might, therefore, with some reason be attributed only to the glands, especially as there was no evidence of middle-ear or mastoid trouble, as subsequently demonstrated at the first operation.

The absence of rigors at any time also tended to negative lateral sinus pyæmia.

The high temperature, the absence of vomiting, and the persistently rapid pulse were points against cerebral abscess, and in favour of meningitis.

The results of lumbar puncture on May 18th appeared more in favour of tuberculous than either septic meningitis or cerebral abscess. The value of this method of diagnosis may now be considered generally.

*Lumbar puncture.*—We come now to consider the value of lumbar puncture, described in 1891 by Quincke, as an aid to diagnosis. The cerebro-spinal fluid is drawn off by puncture between the third and fourth lumbar vertebræ. In infants it is usual to puncture through the space met with at the level of the line joining the highest points of the iliac crests, the patient, as always preferably, lying on the side, with knees well flexed upon the abdomen. It is desirable to note not only the colour, but the pressure of the escaping fluid, some observers using a simple manometer which is readily attached to the cannula on withdrawing the trocar. Besides the number and nature of the cells, and bacteriological examinations of films and cultures, the quantity of albumen (protein) should be estimated.*

*Suppurative meningitis.*—In a typical case the cerebro-spinal fluid thus evacuated is turbid, sometimes of greenish-yellow tint. It escapes under considerable pressure. Coagulation may be so rapid as to prevent cell counts. With regard to the nature of the clot, Farquhar Buzzard† states

* For further details refer to an illustrated article by Heiman in the 1908 'Medical Annual.'

† 'Brit. Med. Journ.,' September 21st, 1907, on "Lumbar Puncture."

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that it is often yellow, whereas in tuberculous meningitis it is translucent or grey, with perhaps a few white flakes, the difference being probably due to the cell elements in each case. Films show numerous leucocytes, in acute cases, chiefly polymorphonuclear neutrophiles, *e. g.*, over 90 per cent ; in chronic cases lymphocytes, *e. g.*, over 60 per cent., and the pyogenic organism concerned (*e. g.*, pneumo- or streptococci), which can be cultivated on the appropriate media. The amount of albumen varies from a faint trace to a heavy deposit. Protein (tested for in Esbach's apparatus, with picric acid) is increased in quantity, and may amount to 4 grains per litre in acute cases, but in the more chronic may not exceed 1 grain, the amount in normal cerebro-spinal fluid being less than ½ grain.* Exceptionally the fluid may be clear, and contain a few lymphocytes only, or neither cells nor organisms.

In the second of the cases of pneumococcic meningitis (Case 6) we have been studying, the fluid obtained by lumbar puncture was clear and limpid, evidently under considerable pressure, but contained no albumen, and only 40 leucocytes, almost entirely lymphocytes. There were no organisms either in film preparations or culture.

In *tuberculous meningitis*, however, the fluid is typically clear or slightly turbid, and though it may present nothing characteristic macroscopically, it may contain two or three hundred leucocytes, lymphocytes markedly predominating over polymorphonuclears. The fluid is said to be generally sterile, but according to Dr. E. H. Shaw, the pathologist who kindly investigated the case under consideration, the finding of tubercle bacilli is largely a question of patient search, and this view is confirmed by Graham Forbes,† who, in his last ten months' record has found tubercle bacilli in 25 out of 31 cases (80 per cent.).

The fluid escapes under considerable pressure. Protein averages rather less than 1 grain per litre, but may amount to 5, or rarely 10 grains per litre. (Rous.)‡

Rous says that "While a diagnosis of tuberculous meningitis, *intra vitam*, cannot be absolute without the discovery of the characteristic bacilli, or a positive result on animal inoculation, yet a clear, or slightly turbid, cerebro-spinal fluid, under increased tension, with over 100 cells per c.mm., amongst which lymphocytes predominate, and an increase in protein under Esbach's test, strongly point to the lesion. A long-standing purulent meningitis, in which the many polymorphonuclears have been replaced by a few mononuclears, and an occasional case of paresis, are the only other conditions likely to yield such a condition. One can affirm the absence of the disease when the fluid conforms to the normal as regards pressure, protein and cell contents."

In *cerebral abscess* the lumbar puncture is of no value, the fluid, typically, being free from pus; but where complicated with meningitis it should resemble that in septic meningitis, being turbid from containing numerous leucocytes, chiefly polymorphonuclears, and the causative organism, *e. g.* streptococcus, which can be cultivated in the usual way.

Of two cases included in Graham Forbes's paper:

(*a*) In one there were only a few degenerated cells. No organisms found. At the autopsy, pus from the right lateral ventricle yielded pure cultures of *Staphylococcus pyogenes aureus.*

(*b*) In the second case the fluid contained a faint haze of albumen, very few leucocytes, no organisims in film, cultures sterile.

Blood count = 18,500 leucocytes per c.mm.

The pus in the temporo-sphenoidal abscess yielded pure cultures of streptococcus.

Further, in the case of a large cerebral abscess in the left frontal lobe, reported by Dr. Langdon Brown,* where no source for the infection could be discovered at the autopsy, lumbar puncture had yielded about 6 drm. of clear, light yellow fluid containing 32 leucocytes per c.mm., almost all lymphocytes. The fluid was sterile, though films and cultivations made from the pus from the cerebral abscess yielded pure cultures of the *Streptococcus pyogenes aureus.*

*In cerebral laceration, or sub-dural hæmorrhage*, the cerebro-spinal fluid is blood-stained.

*In extra-dural hæmorrhage*, from injury to the

* Peyton Rous, 'Amer. Journ. Med. Sci.,' 1907, vol. cxxxiii, "Studies of Cerebro-spinal Fluid, with special reference to Pressure, Protein-content, and Number and Character of the Cells."

† 'Quarterly Journ. Med.,' January, 1908.

‡ *Ibid.*

* "Some Points in Cerebral Localisation," in 'St. Bartholomew's Hospital Journal' June, 1907.

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meningeal artery or a venous sinus, the fluid evacuated is clear.

Ballance* speaks of the great value of lumbar puncture in traumatic lacerations of the cerebral substance when the patient passes into the stage of cerebral irritation. In one such case he withdrew 2½ to 3 ounces of red-stained, cerebro-spinal fluid, and on each occasion quiet sleep was obtained afterwards for four hours, the man making a good recovery.

*The frequency of pneumococcic meningitis.*—Eyre† has recently published the records of the Bacteriological Department of Guy's Hospital for five years (1903–7), enabling us to obtain some idea of the comparative frequency with which the pneumococcus is met with in situations other than pulmonary tissue. There were 168 instances where the presence of the pneumococcus had led to the formation of an exudation possessing all the physical attributes of pus, apart from 7 cases of septicæmia, 2 cases of infective endocarditis, and 24 cases of pleurisy with clear fluid, all due to the same organism.

Dealing with the 168 cases, we may note the following interesting record:

Purulent pneumococcic lesions (for the years 1904–7):

| | Cases. |
|---|---|
| Purulent meningitis | 14 (8 occurring in 1907). |
| Abscess, cerebrum | 2 |
| ,, cerebellum | 1 |
| ,, mastoid | 5 |
| Empyema, pure | 95 |
| ,, mixed | 6 |
| Purulent pericarditis | 9 |
| ,, peritonitis, pure | 10 |
| ,, ,, mixed | 1 |
| Appendicular abscess, pure | 1 |
| Appendicular abscess, mixed | 4 |
| Arthritis | 8 |
| Other cases | 12 |
| Total | 168 |

Graham Forbes‡ gives the results of the bacteriological examination of lumbar puncture fluid at the Hospital for Sick Children in 142 cases occurring in the same three years (1904–7).

Pneumococcic meningitis was thus ascertained to be present in nine instances, in one of which it was secondary to tubercle of the meninges.

From these two sources we see that, at Guy's Hospital, there were at least 14 cases in five years—about an annual average of 3 cases, but the numbers show a steady increase, there being 8 cases in 1907 alone; and, at Great Ormond Street Children's Hospital, another 9 cases were demonstrated in the same years by means of lumbar puncture. Osler's series of 93 cases of meningitis shows that over 22 per cent. were due to the pneumococcus; and Turner, recording in 1907 an investigation of the outbreak of meningitis amongst Rand miners (natives), states that in a series of 70, 17 cases were proved to be due to the pneumococcus (Eyre).

*Route of infection.*—The organism, as I need not tell you, was first isolated from the healthy mouth, and it may be said to be indigenous there, and in the fauces and the nose.

Its chief route of infection, says Eyre, is by way of the respiratory tract; but it has also been proved to enter *viâ* the alimentary canal, giving rise to gastritis, appendicitis, etc. From the lung the cocci find their way readily into the circulation, and are deposited in any area of lowered vitality. This was probably the mode in Case 5, where the meningitis supervened upon pneumonia. This type of infection as a complication of pneumonia, is much more frequent in adults than children (Osler). In Case 6, the meninges appear to have been infected from the thrombosed right lateral sinus, and infection of the sinus certainly followed the attack of tonsillitis, indirectly through the general circulation or, more probably, by direct infection of the lateral sinus, *viâ* certain emissary veins. As is well known, infection of the meninges not infrequently supervenes upon sinus thrombosis.

Apart from the evidence of such a source of infection in this case, it should be stated, however, that local infection of the meninges (from nose, mouth, fauces, etc.) is believed by other observers to spread directly by the lymphatics through the body of the sphenoid, or through the cribriform plate, to the region of the pituitary body, and so to the meninges, and this is the view held by a some-

* "Some Points in the Surgery of the Brain and its Membranes," 'Trans. Med. Soc. Lond.,' 1906.

† Erasmus Wilson Lecture, "The Pyogenetic Activities of the Pneumococcus," 'Lancet,' February 22nd, 1908.

‡ *Ibid.*

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what recent German Commission as regards the route of invasion generally in the case of epidemic cerebro-spinal meningitis.

The two cases of meningitis here recorded (Cases 5 and 6) represent two of the three types of pneumococcic meningitis; the third, primary pneumococcic meningitis, being, according to Osler, much more common in France than in England or the United states, bacteriological examination of the lumbar puncture fluid enabling one to distinguish the Gram-positive *Pneumococcus* from the Gram-negative *Diplococcus intra-cellularis*, causing epidemic cerebro-spinal, and also, probably, posterior basic meningitis.

*Prognosis, diagnosis, and treatment.*—By common consent, hitherto, the prognosis in pneumococcic meningitis has been considered practically always hopeless. The disease is always, or almost always, secondary to some other lesion due to the organism indicated. The first requisite, of course, for any successful treatment is definite and early recognition.

With or without direct clinical evidence, such as meningitis supervening upon pneumonia, or pneumococcic empyema, or by the discovery of the organism in any other pathological effusion, two additional methods are now available. One is the result of Wright and Douglas's epoch-making work, whereby an early diagnosis can in very many cases be made by investigating the opsonic index of the patient's blood-serum for the pneumococcus; the other, lumbar puncture, to which allusion has already been made, gives, in many cases, at least, still more positive assistance, the organism itself being discovered in the cerebro-spinal fluid.

If puncture results are negative, owing probably to meningitic adhesions in the early stages localising the infective process, in the way an appendix abscess may be shut off from the general peritoneum, and failing positive evidence with regard to the opsonic index, where the presumptive evidence of pneumococcic meningitis is strong, it would be quite justifiable in such an otherwise hopeless condition to trephine, and from any exudate detected make a diagnosis and prepare a vaccine (see below). The relief of headache and optic neuritis, of themselves alone, would justify the trephining—a method urged by Sir Victor Horsley, in the case of intra-cranial tumours, even when their removal is out of the question.

Ballance,* indeed, "convinced that our treatment of intra-cranial infection has too long been encrusted with conventionality," advocates free drainage, and says that "in general suppurative meningitis, the operation affording the best chance of success is one which provides a free bilateral opening, and allows the escape of pus from the subarachnoid space. It has been, moreover, rightly suggested that the spinal theca should be opened in the lumbar region to permit irrigation from the cranial to the spinal cavity."

This treatment is still under trial.

*Vaccine treatment of meningitis.* — Returning, however, to the subject of lumbar puncture, it is not only diagnostic, but has the further advantage, in the absence of other sources of effusion containing the organism special to the condition, that a vaccine can also be prepared at the same time from the same fluid, and this is advisable, as vaccines from stock laboratory cultures are not always satisfactory in the treatment of pneumococcus infections elsewhere. If there is already an effusion, as in empyema, containing the organism, the nature of any brain complication can be fairly correctly diagnosed, and a vaccine prepared from the pus, further confirmation, if desired, being arrived at by preliminary lumbar puncture.

This method of vaccine treatment for meningitis has already achieved some success in the epidemic cerebro-spinal form of disease, and since, in experienced hands, it appears devoid of risk, and the prognosis otherwise is so grave, it is certainly worthy of extended trial in other forms of meningitis, due to the pneumococcus and other organisms.

In tuberculous meningitis, supervening upon advanced lung mischief, however, such treatment, I fear, offers no more hope than in the last stages of phthisis itself, but the relief of tension and distressing headache obtained by trephining would seem to encourage more resort to surgical intervention in future, and may perhaps serve to correct some diagnoses.

Vaccine treatment for meningitis, due to the pneumococcus especially, seems to me the most hopeful suggestion, and all the more promising in view of the results recently mentioned by Eyre†

* *Ibid.*

† *Ibid.*

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of cases relating to two other types of lesions due to the pneumococcus. The objection that pneumococcus lesions elsewhere are amenable to treatment not applicable to meningitis does not hold so strongly in the case of vaccines, especially where an early diagnosis is made by the means here indicated.

(1) In a little girl double pneumonia was followed by empyema, on one side on the twentieth day, on the other two days later.

Suppurative arthritis of the right hip was operated on on the thirtieth day, and an iliac abcess opened.

On the fifty-fourth day an abscess in the gluteal muscles behind the great trochanter was incised. The case now looked hopeless. A vaccine prepared from the pneumococcus in the empyema pus was used on the fifty-second day, and the patient was discharged to a convalescent home on the eighty-second day.

(2) A boy under treatment for tuberculous peritonitis, with secondary pneumococcus infection.

The pus, which was crowded with pneumococci, discharged freely through the ruptured umbilicus.

On the twenty-second day after admission to hospital, and again on the thirty-third day, stock pneumococcus vaccine was injected with no effect on temperature or general condition.

Vaccine was then prepared from the particular pneumococcus isolated from the peritoneal pus of the patient, and improvement at once followed an injection of a dose on the forty-fourth day. The patient put on weight, the pus gradually disappeared, and the temperature came down.

It may also be worth recording here that a case of psoas abscess under my care, with persistent sinus for eighteen months after operation by another surgeon, at once closed after three injections of tuberculin.

*The use of serums.*—It may have occurred to you to say, why not use anti-pneumococcic serum? Being purely bactericidal, and not antitoxic, Eyre, an acknowledged authority, says that it is practically of no service where a profound toxæmia already exists by the time there are sufficient data upon which to make a diagnosis, which is surely the condition in pneumococcic meningitis.

*Repeated lumbar puncture in the treatment of meningitis.*—Mr. Barker has quite recently* reported three cases, with two recoveries, where the method of repeated lumbar puncture (in one case performed twelve or thirteen times) was employed in either meningitis or cerebral abscess, or both, following otitis media and mastoiditis.

Of the successful cases, the fluid in one contained the *Micrococcus catarrhalis*, the other case containing a diphtheroid organism and a staphylococcus.

In the unsuccessful case the fluid contained a virulent streptococcus. In the case of this organism, and of the tubercle bacillus, Barker says repeated tappings seem to be of little or no use.

In the subsequent discussion, Ballance, dividing meningitis into serous and suppurative types (serous meningitis being often seen in association with acute spreading otitis), considered that: "If the suppurative stage could be averted by combined local and operative measures, with lumbar tappings, it would be of great importance. The fulminating type of meningitis did not yield to tappings."

As regards tuberculous meningitis, however, let me quote to you G. Reabold,* who reports the successful result of twenty-four tappings in a case confirmed to be such, bacteriologically, and from which, in all, 674 cc. of cerebro-spinal fluid were evacuated. A steady and almost immediate improvement and cure followed, in spite of the tendency to rapid reaccumulation of the fluid in the intervals. The consequent rise of intra-cerebral pressure was manifested by headache, the state of which served as an indication for further puncture. As the fever and amount of albumen present in the cerebro-spinal fluid diminish the intervals between the punctures may be lessened.

As regards repeated puncture in this way, when performed with due care and the strictest antiseptic precautions, there is considerable evidence that relief of symptoms, if only temporary, results, and in some cases definite cure follows.

To summarise recent suggestions for the treatment of these otherwise generally fatal cases, these are as follows:

(1) Early bilateral drainage of the skull, perhaps with counter-opening of the lumbar spinal canal, to permit of continuous irrigation (Ballance).

(2) Repeated lumbar puncture (Barker, etc.).

(3) (The author's suggestion.) The use of appropriate vaccines, combined with lumbar puncture where necessary, either for diagnosis, or to obtain material for the vaccine, or for the relief of urgent pressure or other symptoms.

Where the indication otherwise is clear, if lumbar puncture is negative, trephining would be quite justifiable, with a view to confirming the diagnosis, relieving the urgent symptoms, and securing material for vaccine treatment, if not otherwise obtainable.

With more active attempts at treatment of this kind we may, in conclusion, hopefully anticipate a steady reduction in the mortality from meningitis, and so also vastly improve the prognosis in other diseases in which, so often, it has hitherto proved the fatal complication.

*May 25th*, 1908.

* 'Royal Society of Medicine, Section of Surgery,' March 10th, 1908. (See also 'Brit. Med. Journ.,' March 21st, 1908.)

* 'Münch. med. Woch.,' 1906, p. 1709. (See also 'Amer. Journ. Med. Sci.,' vol. cxxxiii, 1907.)

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# THE CLINICAL JOURNAL,

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## A CLINICAL LECTURE

ON THE

# SPREAD OF MALIGNANT DISEASE TO THE LYMPHATICS OF THE GROIN.

Delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—Some time ago I lectured to you on malignant infection in the lymphatic glands of the neck, on a later occasion on the spread of malignant disease to the lymphatics in the axilla, and to-day I shall lecture on the spread of malignant disease to the lymphatic glands of the groin. You will again observe that I have made free use of Professor Poirier's admirable work in the lymphatics and which has been so ably translated by Mr. Cecil Leaf. I have chosen this subject, first, because it is a difficult one, and secondly, because unless you have a competent knowlege of the lymphatic glands in the groin, and of the manner in which malignant disease spreads to them, you will not fulfil an essential condition in operations upon malignant disease; and that essential condition is, that when the operation is begun you should not cease until all that malignant disease has been removed. In the cases to which I shall allude the disease spread to the glands in the groin from two main sources: first, from cancer of the external organs of generation of the female and of the male, and in one instance from cancer of the bulbous urethra; and next the disease spread to the lymphatic glands of the groin from sarcomas of the thigh. But, in addition, there is one case of tubercle of the lymphatic glands of the groin in which the tubercle had spread from hypertrophic lupus of the thigh and of the nates; and another in which a cancer of the skin of the thigh was in

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question, the inguinal glands being enlarged but not cancerous. In considering the cases we will proceed, as we invariably do, in a due anatomical order. I will, therefore, take the original disease first, and see how the disease passes from the primary growth along the lymphatics into the glands, and then trace its spread in the groups of glands. Thus we shall find that we have to consider malignant disease in the superficial inguinal glands, then malignant disease in the intermediate group of glands situated in the saphenous opening amongst the cribriform fascia, and next malignant disease in the deep inguinal lymphatic glands, situated by the side of the femoral vessels; and, lastly, malignant disease of the glands within the pelvis. With reference to the original source of infection, you might suppose that it was easy to diagnose that. But difficulties arise. It is easy for you to understand how a small, hard lump, which is followed by ulceration, and which is situated on the external organs of generation, is easily mistaken for a venereal sore. Perhaps the age of the patient and the history ought to put you upon your guard. Perhaps they would, but there is another thing which ought to put you more upon your guard. Those who have acquired the habit of making an examination of the whole patient would observe the absence of the usual anæmia of syphilis, the absence of local and of general lymphatic engorgement, and the absence of syphilitic eruptions on other parts of the body. However this may be, the clinical diagnosis may be difficult, and I may at once say that the histological diagnosis may likewise be difficult. I recall very vividly the case of an unfortunate woman who had a small ulcer in the vestibule at the side of the urethra. After an anæsthetic had been given an immediate histological examination was made, and the histologist pronounced the base of the ulcer to be inflammatory. But I observed amidst the cells, which had the appearance of inflammatory cells, others of an epithelial type, and made up my mind that it was a rapidly-growing carcinoma, and proceeded there and then to do a sweeping operation. When the carcinoma is of that type the outlook for the patient is grave, and within but a brief space the disease returned at its original site and also in the glands of the groin. The whole was again removed, but it was not long before extensive recurrence took place and led to a fatal end. I also recall the case of a man in whom, after a history of four months, an induration could be felt but could not be seen, because he had phimosis. Here, again, difficulties in diagnosis arose. But a small nodule was removed for histological examination, and found to be carcinoma. After the epithelioma underneath the prepuce had been exposed it was still difficult to diagnose, for it was of that warty variety, in which there is more papilloma than ulceration. It might very reasonably have been mistaken for an innocent papilloma of the glans and prepuce. I mention this to you particularly, because if you are about to make a microscopical examination under such circumstances, it is of extreme importance that you should choose a part of the growth near its base, so as to ascertain whether it has those epithelial rootlets, which are so typical of carcinoma, growing into the deeper tissues, and which, by-the-by, indicate that the carcinoma is beginning to creep along the lymphatic channels. That spread along the lymphatic channels is sure to occur, and it is sure to occur quickly. And we are now proceeding to the next step, and that is to consider the manner in which it spreads. In the first place it spreads from the organs of generation towards the symphysis pubis. And here let me draw a distinction which is of some importance—the division of lymphatics into superficial and deep. The superficial lymphatics are those above or superficial to deep fascia in all parts of the body; the deep lymphatics are those beneath it. These carcinomata upon the prepuce and upon the skin of the penis, upon the labia majora or minora, spread along the superficial lymphatics until they come to the symphysis pubis. And here you have to recollect that the lymphatics do not of necessity pass up the side upon which the original growth is. They may cross the pubic symphysis in three ways: First of all, the lymphatics may cross bodily from one side of the body to the other, or, when they reach the pubes, they may bifurcate and pass to both sides, or they may form an intercommunicating plexus in front of the pubes. This is not a matter of mere anatomical interest, but one of clinical importance, because if clear upon this point, when you see carcinoma, or a suspicious sore, or chancre, upon one side of the organs of generation, you will not rest satisfied until you have examined the inguinal glands, not in one groin, but in both. When the lymphatics have reached

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the pubic symphysis, they then pass outwards as satellites of the superficial external pudic vessels into a group of glands which is situated in Scarpa's triangle, the superficial inguinal glands. The superficial inguinal glands (Fig. 1) are arranged in a kind of ring round the boundaries of Scarpa's triangle. Some rather large ones are situated at the base of Scarpa's triangle, parallel to the superficial crural arch. I hardly think it is necessary to separate this or any other group of glands in Scarpa's triangle into sets, for reasons which I shall explain to you presently. Other glands lie along the outer boundary of Scarpa's triangle, and they may reach a long way towards the anterior superior spine of the ilium. So that sometimes I have removed enlarged glands from close up to the attachment of the sartorius. This point has a bearing on the question of skin incisions. Next, the inguinal lymphatic glands are met with below, at the apex of Scarpa's triangle, and these are some of the biggest.

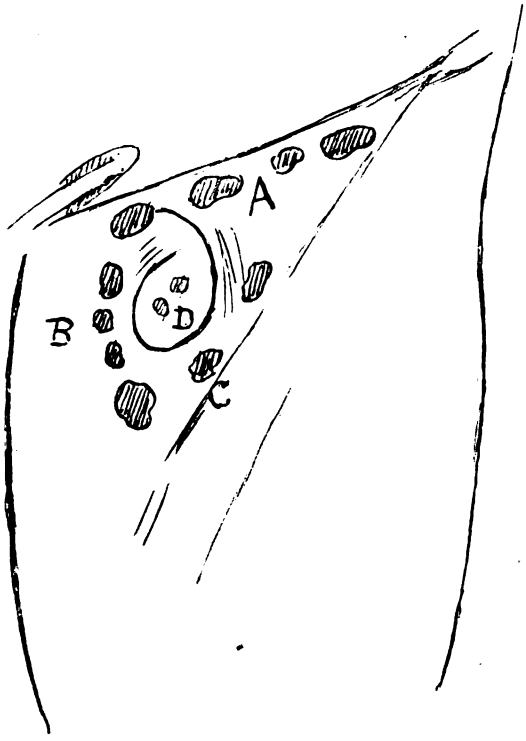


FIG. 1.—Superficial inguinal lymphatics grouped round Scarpa's triangle. A. Superior group. B. Internal. C. Inferior. D. Glands in cribriform fascia. Intermediate.

I will now return to the division of the superficial inguinal glands into groups. This refinement is hardly needed. Disease which begins at a certain point and spreads into the inguinal lymphatics, does not necessarily spread into the nearest group of lymphatic glands. Thus, cancer from a growth upon the front part of the labium ought to pass outwards with the superficial external pudic artery, and pass into the group of glands which lies inside the femoral vessels at the inner end of the crural arch. But they may pass straight into the lower group of glands. Therefore, I would ask you not to overlook the lower group of glands, or the cancer may miss both the superior and the inferior, and pass straight outwards into the outer group of glands; so anyone who began to operate with preconceived ideas might overlook enlarged glands. Again, the intercommunication between these various groups of glands is so free that shortly after one has become cancerous or infected the condition spreads to the others. Again, the various forms of infection do not follow proper physiological routes. I think there is much evidence to show that cancer may spread along lymphatics against the lymph stream, and against the valves, so with this in our minds it is scarcely necessary to refine too much upon the groups of lymphatic glands in Scarpa's triangle. The following illustrates the manner in which carcinoma spreads, and the steps of the operation for its removal:

In a case of carcinoma of the organs of generation, which was in Lucas Ward but a few months ago, the carcinoma spread along the superficial lymphatics into the superficial inguinal lymphatic glands, which could be felt. Therefore, after the original growth had been taken away the second stage of the operation was directed towards their removal. Now comes the question of the best skin incision to expose these superficial inguinal lymphatic glands. You must already have gathered that it must lay bare the whole of Scarpa's triangle, because it is necessary to search for and remove glands which are (1) at the upper and inner side, (2) the glands at the apex, and, as I have mentioned, (3) glands upwards and outwards near the anterior superior spine. I have made various incisions to expose the glands in Scarpa's triangle, and I have come to this conclusion—a very definite conclusion: that those with pointed angles are by no means satisfactory. It is undesirable that any incision in Scarpa's triangle should be carried towards the pudenda. The reason is that it is so difficult to preserve an incision in that region from infection,

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so that in making triangular flaps I have made them with the base of attachment towards the pudenda, and with the apex outwards. But the whole of those triangular flaps often became livid and threatened to die. And over and over again the apex of those pointed flaps has died. Perhaps you will be inclined to say that that does not mean very much. But as long as the wound is unhealed the patient is uneasy, and cannot be considered well. Also, the area which had to granulate and heal over is apt to ulcerate again. So I cannot think that pointed flaps are satisfactory. Therefore, in my later cases I have exposed the inguinal glands by a semilunar flap. When a semilunar flap has been made, it is a matter of dissection and of knowledge of the distribution of the inguinal glands to effect their complete removal. The surgical difficulties are of the most trifling description.

But now we come to another point. In the saphenous opening amidst the meshes of the cribriform fascia there are additional lymphatic glands, which I will call intermediate, and the cancer speedily spreads from the superficial into the intermediate group and so on into the deep. Hence it is necessary to remove the cribriform fascia and also that of Scarpa's triangle, and expose thoroughly the femoral vessels and their sheath. I have just said that the deep lymphatics get infected from the superficial, but that is not the whole truth. In cancer of the glans penis, in cancer of the clitoris, and in cancer of the bulbous urethra, the infection runs along the deep lymphatics towards the pubes, and thence onwards as satellites of the deep external pudic vessels, in their course beneath the deep fascia, into the deep inguinal glands (Fig. 2). I believe, also, that in the case of cancer of the bulbous urethra the disease went that way; but clinical evidence is oft-times wanting in scientific precision. A man had had a urethral stricture for many years. It was kept from closing by the passage of instruments. But in May, 1904, he got retention. I performed internal urethrotomy, and divided a stricture in the bulbous urethra measuring 8 mm. When I had finished a 30 mm. bougie passed with ease. Four months later he returned with an impassable stricture. When external urethrotomy was done the urethra was found imbedded in a hard mass, which looked like scirrhus. This had been felt before the operation, and its suspicious nature had been surmised, but owing, perhaps, to the unusual circumstances, both Dr. Andrewes and myself did not like to commit ourselves upon the result of the immediate histological examinations, especially as some pus escaped from the urethra. Dr. Andrewes, however, afterwards diagnosed carcinoma, and in my absence Mr. Harmer very kindly performed the operation. This consisted in the removal of the organs of generation, together with the inguinal

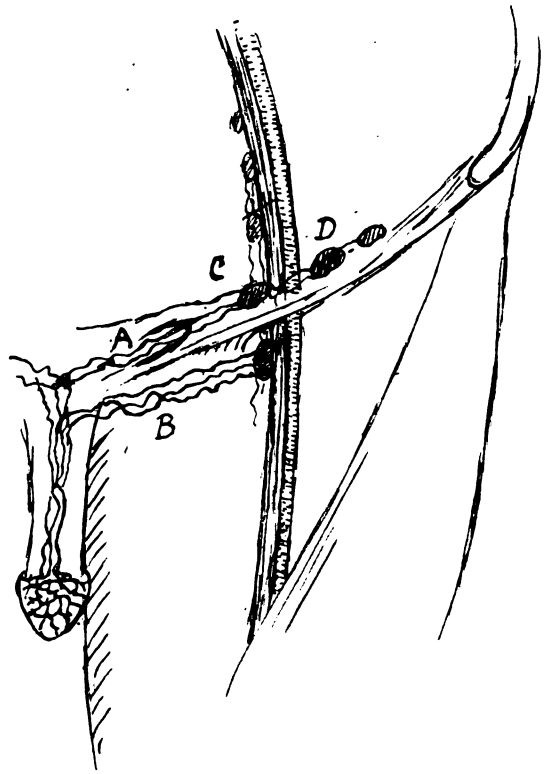


Fig. 2.—Deep lymphatics. A. Deep lymphatics entering external abdominal ring to enter D. D. External retro-crural gland. B. Deep lymphatics entering deep inguinal gland in femoral canal and passing onwards into C. C. Internal retro-crural canal.

glands, which were enlarged. It is to be regretted that no post-mortem examination was made, and we have no record of the histology of the glands.

When the deep lymphatics reach the pubes they then spread outwards in two directions; some following the crural route pass outwards as satellites of the deep external pudic artery, underneath the pectineal fascia, and pass to the deep glands close to the femoral artery (Fig. 2). There is another route, the inguinal, which consists of a few lymphatics,

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which run into the inguinal canal underneath the spermatic cord, and enter the outermost of the retrocrural group of the pelvic glands (Fig. 2). I commend the lymphatics which run up the inguinal canal to your attention, because they form, what I have called on other occasions, a short-circuiting of the lymphatics of the infected area into the pelvic lymphatics, all the superficial and deep inguinal being missed. The removal of the fascia lata covering Scarpa's triangle exposes the deep lymphatics by the side of the femoral vessels.

Now comes another and important stage of the operation, and that is to explore the femoral canal and ascertain the condition of the glands which are in it. In the femoral canal there are usually one or two glands. The gland below may be of some considerable size. But there is another gland which is situated at the mouth of the femoral canal, and called in books on anatomy the gland of Cloquet. And it is said that Cloquet's gland may become inflamed and enlarged, and be mistaken for a femoral hernia. But to my mind one of the chief interests of that gland is not that. My interest in the gland of Cloquet is rather this: Supposing there is some sort of anatomical barrier between one group of glands and another, clinical experience teaches that malignant disease does not easily pass from one group to the other. But if there is not a satisfactory barrier it passes easily. And in this particular instance the gland of Cloquet is situated in a very delicate piece of tissue, the septum crurale, and is intermediate between the deep inguinal glands and those inside the pelvis.

Why did I say that we ought to explore the femoral canal? During the course of the operation I felt the femoral canal, but could feel nothing in it. But, distrustful of the sense of touch I proceeded to look, and then found enlarged lymphatic glands in the femoral canal. Now, one of these enlarged lymphatic glands, Cloquet's, is almost within the pelvis, and its enlargement is the clue to the next step in the operation. Clearly I was not at all likely to relieve the patient of malignant disease unless I proceeded further and explored the pelvis, and if necessary removed the retro-crural glands. You may think of these retro-crural glands very much as you have been thinking already of the superficial inguinal glands. There is a row of superficial inguinal glands parallel to the lower edge of the superficial crural arch. There is a similar group of glands behind the crural arch lying in the sub-peritoneal tissue, and called the retro-crural glands. They are sometimes divided into two groups: one situated just outside the external iliac artery, and therefore, very close to the internal abdominal ring. That is the group which receives the deep lymphatics which pass up the inguinal canal (Fig. 2). There is another group, situated behind the inner end of the crural arch. This receives vessels from the femoral canal, the pelvis, and from the abdominal wall. I made an observation about this internal group of crural glands which I have not seen stated by the systematic anatomist, namely, that lymphatics pass behind the pubes and open into the group of the opposite side. The occasion for observing that was in a young woman who had an acute appendicular abscess behind the inner end of the right crural arch. It was opened and treated in the usual manner. But during convalescence she had inflammation of the retro-crural glands on the left side, so we thought for some time that they, too, were going to suppurate.

There is another remark about these retro-crural glands that I want to make to you: I have said nothing about the "water-sheds" of the inguinal glands, because that does not appertain to the present subject. But the superficial crural glands have an enormous water-shed and receive lymph from the organs of generation, from the skin of the buttocks, and from the lower part of the abdomen, as high as the umbilicus. The deep crural glands receive lymph from the back of the abdominal wall, from the back of the umbilicus. Not long since, a patient in Harley Ward, who had a cancerous growth of the umbilicus, had glands which could be felt behind the inner end of the crural arch, no doubt carried along the lymphatics into his internal and external retro-crural glands. How are you to get at those retro-crural glands? On two occasions, first of all in the female to whom I am referring, and next in the case of a man with sarcoma of the thigh to whom I shall presently refer again, the skin flap was lifted up towards the abdomen. In the man it was a semilunar flap, which was most convenient for the purpose. Then I proceeded to operate as if about to tie the external iliac artery. The fibres of the external oblique were split, and afterwards those of the internal oblique and transversalis. After

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the fascia transversalis had been cut through the glands were easily exposed. These lie in the loose sub-peritoneal connective tissue, and are very easily removed. They have very few attachments and present no anatomical difficulties. After the retro-crural and also some external iliac glands had been removed, the abdominal wall was repaired in the usual manner, layer by layer. You naturally inquire what has been the result as regards ventral hernia? A man occasionally comes who has had this operation performed a year ago. Although we have examined him on several occasions, and although he has performed rather laborious work, there is no trace of any bulging. And, therefore, if you do have occasion to open the iliac fossa you may make up your minds that if it is done in a proper manner there is no danger of ventral hernia.

Now, I have taken you step by step from the growth upon the pudenda as it passes along the lymphatics into the superficial and deep inguinal glands, and then into the pelvic. I have told you the best skin incision, and how Cloquet's gland should be looked for. And, lastly, I have discussed the manner in which the pelvic glands should be approached.

I will now proceed to the next group of cases, that in which the disease had spread to the inguinal lymphatic glands in cases of sarcoma of the thigh. Here, again, I would point out that the original diagnosis may be by no means easy. One unfortunate man who was under our care began, two years before I saw him, by having a small tumour removed from the subcutaneous tissue of his thigh. It was small, encapsulated, and to the naked eye it looked like fat. Within a very short time he had others, which were removed, and found to be myxo-sarcoma. Then, at intervals of about four months, others appeared, and yet more at intervals of about ten months. When he came under my care he had a growth in the scar of the old operation, and in addition enlargement of the glands in Scarpa's triangle, and of glands in the iliac fossa. The enlargement of the glands in the iliac fossa was felt whilst he was under an anæsthetic. You must be exceedingly cautious how you say you feel anything within the abdomen. Statements made without an examination under an anæsthetic are often open to doubt. But as these enlarged glands were felt an operation was performed similar to that in the case of the female of whom I have just been speaking. It consisted of these stages: First of all the myxo-sarcoma was removed locally. Next, a semilunar flap was turned up from Scarpa's triangle, and all the deep and superficial lymphatics were removed. Then the iliac fossa was opened by an incision such as is made for ligation of the external iliac artery. We found numerous enlarged lymphatic glands, which were all removed. There is this to be said: that I believe pathologists have reported that no sarcoma was to be found in these enlarged lymphatic glands. To my mind that is a little puzzling, because I know from clinical experience that when they are not taken away they continue to grow; so I cannot help suspecting that their enlargement is actually due to the growth of sarcoma in them. I cannot help thinking that the pathologists have not yet succeeded in obtaining a stain to enable them to differentiate the growth of sarcoma from the lymphoid tissue of the glands themselves. I had another occasion for observing the difficulty in diagnosing these tumours of the thigh. Five or six years ago a man had an ossifying chondroma growing from the front of the left great trochanter. When this was removed it had a thin, bony stalk, and a cap of partly ossified cartilage. You would say that this was an innocent tumour. Whatever the term "innocent" may mean, I suspect that that often depends upon the innocency of the mind. In four years' time this man came back with a large tumour at the site of the previous operation, and enlargement of the superficial inguinal glands. I proceeded to amputate at the hip in the usual way, and now the usual way to amputate at the hip is this: A longitudinal incision is made along the course of the common and superficial femoral vessels. That is carried to the junction of the middle and upper third of the femur. The skin flap is lifted up, and it enables you to proceed with the first step, namely, the removal of the enlarged glands. The point of interest to me in these enlarged glands was this—that those near the tumour contained chondro-sarcoma. But as we proceeded towards the femoral vessels and towards the deep lymphatics they ceased to have chondro-sarcoma in them, so that I did not expect to find any sarcomatous glands within the pelvis. After the glands had been removed, the next stage of the operation was to tie the common femoral artery and

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vein. For obvious reasons the artery was tied first and the limb emptied of blood, and then the vein was tied. In the next stage the femoral canal was explored, to see whether the deep lymphatics contained sarcoma, but they did not. I need not proceed to mention the subsequent stages of amputation of the hip: I will merely say that it is best to proceed very slowly. Tie the femoral vessels as a beginning, and then go quietly and slowly on, dividing the muscles one by one; when a vessel bleeds clip it, or if that is not sufficient, tie it. And so you can go on, and by the time the operation is at an end you need not have caused the patient to lose more than ten ounces of blood. You may say that the prolongation of the operation will result in greatly increased shock. I have not observed that. During the hurried operations of earlier days the patients used to lose a great deal of blood, and often died of shock. With regard to these cases of infection of the glands by sarcoma of the femur, I will ask you to take your minds one step further. I have only spoken of infection of the superficial and deep inguinal glands in sarcoma of the thigh. But obviously disease may spread from the deep inguinal lymphatics into those in the iliac fossa, and that has been ascertained more than once in cases upon which I have performed amputation of the hip. I merely wish to say that should there be the slightest suspicion in your minds, after exploring the inguinal glands, and after exploring the glands in the femoral canal, that the retro-crural or iliac glands may have become infected, it is easy to open the iliac fossa and to remove those glands. You may imagine that after such a severe operation as amputation of the hip it would be impolitic to continue to perform the further operation of removing the glands in the iliac fossa. But I have already said that the glands in the iliac fossa lie in very loose tissue, and have very few attachments, and that they are very easily separated from their surroundings. Therefore, according to my observation, it is hardly any addition to the severity of the operation to continue and remove them.

Before ceasing I will mention one other case, which is interesting. A man was in Harley Ward who had, many years before, been burnt. The area of his burn had remained an open sore and refused to heal. When he came under our care we guessed that that burn might have become epitheliomatous. So the burn was removed, and was there and then found to be epitheliomatous. He had also a number of enlarged glands in Scarpa's triangle, and we therefore proceeded to remove the glands. But although it is easy to see carcinoma in enlarged glands, we could see none in these. So you must not assume that every enlarged gland is therefore a malignant gland. The meaning of the enlargement in these cases is, that there had been a continual absorption of some kind of sepsis from the ancient wound, which had become epitheliomatous, and that the glands were chronically inflamed. Nevertheless, if a person has, in association with any growth, such as carcinoma or sarcoma, any enlargement of the lymphatic glands, you must not on any account be content until the whole of those glands have been removed, and until the whole of them have been properly examined. It is a long and difficult subject, and I cannot conclude it with a proper peroration, and tell you that the cases about which I have been speaking have done admirably, and yielded most satisfactory results. I regret to say that, with few exceptions, they have been of the most unsatisfactory nature. I do not attribute that to delay, or to the usual assumption that they were not seen soon enough. But I am sure of this, that the disease has a great deal to do with it, and that many of these people had most dire and dreadful diseases. Some have been satisfactory. In conclusion I want to thank Mr. Trapnell for the very great trouble he has taken to look up the notes of these cases, and for writing to patients to know whether they were still alive.

*June 1st*, 1908.

# THE PROGNOSIS AND TREATMENT OF ACUTE ANTERIOR POLIOMYELITIS.*

By J. S. RISIEN RUSSELL, M.D., F.R.C.P.,
Physician to University College Hospital.

AND DISCUSSION THEREON BY

Dr. C. E. Beevor. Mr. R. Jones.
Mr. A. H. Tubby. Dr. E. F. Buzzard.
Dr. L. G. Guthrie. Dr. F. J. Poynton.
Dr. F. E. Batten. Dr. J. K. Fowler.

Dr. J. S. Risien Russell said that in any attempt to estimate the prognosis of acute poliomyelitis it was necessary to exclude especially Landry's paralysis and acute toxic polyneuritis. The former of these was an exceedingly fatal disease, whereas

* Delivered before the Medical Society of London, with Dr. J. K. Fowler in the chair.

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poliomyelitis was not, and in the latter a degree of recovery might be expected which was not met with in poliomyelitis. Even allowing for the possibility that some cases of acute poliomyelitis died in the febrile stage of the illness, before the real nature of the condition was recognised, the malady was not a fatal one. But it was possible that cases were sometimes overlooked, patients suffering from some febrile malady which was diagnosed as one of the specific fevers, but in which the condition proved, when the patient was able to get out of bed, to have been a case of poliomyelitis. No true estimate of the prognosis of a case of poliomyelitis could be formed until the paralysis was arrested in its progress for at least twenty-four to forty-eight hours, especially when there was any paralysis of the muscles of respiration. Good recovery from the paralysis was not to expected in the majority of instances, although notable exceptions to this rule were to be found. In attempting to form an estimate as to whether recovery was or was not to be expected, the more rapidly power commenced to return in the affected limbs the better was the outlook for the future. An attempt might be made by means of electricity to estimate whether or not the muscles would return to power. A muscle which still responded to faradism after an interval of three weeks from the onset of the attack was one in which return of good power was to be expected. But on the other hand it could not be said that because a muscle did not respond to faradism at the end of three weeks that therefore it would not ultimately recover, because such a muscle might recover to a considerable extent under appropriate treatment. Faradic irritability had been known to return in some instances at as long a period afterwards as twelve months from the onset of the poliomyelitis. From time to time cases were met with in practice in which individuals in after life presented themselves for examination for some other purpose, or for some added nervous malady, such as that which he would refer to, in which a generally diminutive limb was met with, showing uniform want of development in all its muscles, and in which no one muscle or group of muscles could be said to be paralysed or atrophied in excess of the want of development which obtained in the limb as a whole. Those cases were difficult to explain if they were not instances of poliomyelitis earlier in life, in which the condition had recovered and in which the attack had been slight. In those cases there was no actual paralysis, no one movement which could not be performed, and yet there was that general lack of development, shortening, etc., of the limb, such as one was so familiar with in ordinary cases of poliomyelitis in which there happened to be paralysis of individual groups of muscles and atrophy of them. The case might present itself in consequence of some affection of the nervous system, notably where there was some added progressive muscular atrophy. But that condition must be rare as seen by the collection of cases comparatively recently recorded by Potts, who was only able to learn of thirty-seven cases in the literature on the subject. With regard to acute anterior poliomyelitis in adults, there was, in reviewing that subject in literature, great difficulty in eliciting any very definite information as to the prognosis in such cases. He hoped those who would speak would give their experience in regard to the prognosis of the condition in adults as compared with that in children. His experience was that while the condition was infinitely more rare in adults than in children, all the cases in adults which he had chanced to meet had been particularly severe, and although not terminating in death, had resulted in the more severe forms of paralysis such as were met with in children, and the degree of recovery had been much less satisfactory than in many children. Poliomyelitis left, in the majority of cases, effects which were most disappointing, though marked exceptions to that rule occurred. As to whether the cases in which fever existed presented a better prognosis than those in which there was no fever he must leave to the experience of others. His own experience did not permit of any conclusions on that matter. And he did not wish to introduce into the discussion the question of pathology, as prognosis and treatment would provide ample material for one evening's discussion. He was particularly anxious not to do so as two rivals in regard to the pathology of the condition who were present in the room would probably occupy the whole time.

With regard to treatment of an acute anterior poliomyelitis his view was that in the early acute stage of the affection the less that was done the better it was for the patients. They should have absolute rest in bed with, of course, warmth, with the skin induced to act freely, and the bowels kept open.

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The fever which might be present was not sufficient to merit any active interference, with the exception of perhaps some mild febrifuge. But pain was a symptom which demanded attention, and it might be very severe. Local applications might be sufficient to relieve the pain, such as local warmth or leeches to the spine. He was doubtful of the efficacy of counter-irritation of a more active kind. Where local measures failed to relieve pain it might be necessary to fall back upon morphia, which could be given with impunity provided the muscles of respiration were not affected. As to whether any special posture was likely to be helpful was a question influenced by pathology. According to one view it was desirable to keep the patient off his back and have him in the prone position; but on the other hand, the degree of discomfort and irritability generally resulting from such a position did not, in his opinion, justify it, and according to one of the views of pathology it did not matter what the position was. With regard to the question of what active treatment should be employed in regard to the paralysis and when such active treatment should be commenced, there could be no doubt that the most important feature in the therapeutics of the malady in its earlier stages, after the acute febrile condition was over, was massage, for it was the treatment upon which most reliance could be placed in the management of the paralysis. Massage might reasonably be commenced within ten days or a fortnight of the onset of the acute condition. He had seen many cases do quite well in which massage was commenced as early as seven days after the onset, in which there had been no febrile reaction and in which there was no pain to prevent the massage being carried out. But ten days or a fortnight might be too soon in cases where pain was still experienced on any attempt at movement or manipulation. There should also be passive movements, and when the return of power was sufficient to permit of it, resisted exercises as aids in the treatment by massage. In that connection he could not lay too much stress on the encouragement which should be given to children or adults suffering from the affection to perform any voluntary action in connection with muscles which were recovering from the paralysis they had sustained. Such action had much to do with the restoration of function of the muscles. He asked the members to give a pronouncement on the point as to whether skilled massage was desirable in all such cases, rather than that done by unskilled nurses or the mother of the patient. By submitting cases to amateur rubbers, medical men played into the hands of people in this country who practised massage under the title of doctor, but who were not qualified medical men. To oppose such a practice would be to the advantage of the patient and to the benefit of the profession generally. He took the position that galvanism was of value in poliomyelitis, although that also was contrary to the views of certain authorities. Galvanism might be employed through the medium of the bath given to children at a time when the acuteness of the pain prevented massage from being adopted, and it facilitated what was being aimed at, namely, the maintenance of the nutrition of the muscles until such a time as the nerve-cells in the ventral horns of the cord which had not been been destroyed, but were temporarily put out of action, had recovered. With regard to drugs, strychnia had a reputation in the treatment, though whether it was justified he could not say. It could do no harm and was worth a trial. Otherwise drugs helped in so far as they improved the general condition of the patient, and not by virtue of any specific action on the disease. With regard to the surgery of the condition, no one was a greater admirer of the surgeon than himself in view of the advances surgery had made in the treatment of the affection. But he submitted that the disease was one which should be treated on certain defined lines by the physician before the case was handed over to the surgeon. By massage, galvanism and other active measures, including passive movements and resisted exercises, great good could be achieved, and it would be premature to hand over a case for surgical intervention until those measures had had their full trial. The question as to when that treatment should cease and surgery undertaken was a matter for discussion, and he hoped some conclusions would be arrived at on the point from the discussion. The ordinary period for improvement to occur, namely twelve months, was not sufficient, in many cases, as some cases were certainly capable of more improvement after twelve months. The difficulty of long-continued treatment being carried out in hospital cases was recognised, but usually in private cases there was no such difficulty, and the treatment could be

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persevered in for long periods of time. Nevertheless, he warned physicians not to be too conservative if they hoped to retain poliomyelitis in their jurisdiction. The physician should recognise when his resources have come to an end and when the surgeon should be called in, and the physician should take care not to hand over the case to the surgeon with any deformities developed which could have been prevented. In regard to the prevention of deformities it was hoped that more would be heard from the surgeons present, but he could not allow that part of the subject to pass without expressing the strong belief that all cumbersome and weighty instruments were best avoided, and the appliances used should be light and of such a character that they could be removed to permit of exercises which were necessary being carried out. He would not enter upon the various surgical methods which were of value in the condition. Personally, he would suppose that of necessity the cases which permitted of intervention in so far as nerve grafting was concerned must be few as compared with those in which transplantation of tendons could do good, and that to do good in the case of nerve grafting the treatment must be employed earlier than would be necessary in the cases in which transplantation of tendons seemed indicated. Finally, he would be glad to have the authority of the surgeons as to when resection of a joint should be employed with the view of obtaining fixation, and as to what age a patient suffering from anterior poliomyelitis should be subjected to that operation.

Dr. C. E. Beevor congratulated Dr. Risien Russell on the able manner in which he had opened the discussion. He found it difficult to discuss the paper inasmuch as he was in agreement with practically all its conclusions. The diagnosis of the condition was very important, because one was very liable to mistake cases of poliomyelitis for one of the acute specific fevers. Not long ago he saw a young man, æt. 18 years, still attending school, with an attack of fever, which was thought to be influenza. Afterwards he had affection of various muscles, which were paralysed: the diaphragm and the ocular muscles were affected and also the trapezius and serratus magnus. It was regarded as a case of polyneuritis following influenza, but he was now inclined to think it must have been poliomyelitis occurring in an adult. There was no anæsthesia, and it ran very much the course of a case of acute poliomyelitis. He thought electrical testing was a great aid to diagnosis. Dr. Russell had said that if a muscle reacted to faradisation three weeks or a month after the onset the prognosis was very good, and the chances were that the muscle would recover. But if a muscle reacted even twelve months after the onset there was always hope, as Dr. Russell had said. It was only when the muscle had ceased to respond to the galvanic current that there was reason to regard the muscle as hopeless for recovery. With regard to progressive muscular atrophy coming on in a case of infantile paralysis, he had at present under care at the Great Northern Hospital a man, æt. 21 years, who in childhood had infantile paralysis, which affected one leg considerably. That leg was completely wasted. And in the other leg there was some atrophy of the muscles of the foot. During the last few months he had begun to have atrophy of the calf muscles in the good leg. He had not yet had time to test the electrical reactions, and it might possibly be a case similar to those Dr. Russell referred to. Besides the foot muscles, the only muscles affected on that side were the gastrocnemius and soleus. The cases which occurred in adults were very severe, but he did not know whether they were more severe than in children. Many people, by virtue of the name infantile paralysis, did not realise that it could occur in the adult. He did not think the prognosis in cases in which there was definite fever was either better or worse than the others. He agreed as to the importance of rest and warmth. Counter-irritation he did not believe did any good. As to posture, that which was most comfortable for the patient was the best in his view. It was often mentioned in books that the prone position was the best, because of the relief it gave to the cord. The treatment to be most depended on was massage, resisted movements, voluntary movements, and passive movements. Galvanism he regarded as a good thing; it not only kept the muscles in an active condition and prevented them from wasting, but it improved the circulation in the limb and did good in that way. He also agreed that general tonics were best for the disease. Surgical intervention should not be called in until the muscle appeared to be beyond hope of recovery in the

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ordinary acceptation of the term, when transplantation of tendons could be tried. He was sure it was a mistake to operate too soon, because there was always a chance of the muscle recovering. It was very important to be on the look-out for deformities. When the anterior tibial muscles were paralysed the calf muscles contracted with the patient lying in bed, and the foot tended to take a line with the leg. That should be guarded against, either by an elastic arrangement or in other ways.

Mr. A. H. TUBBY said he felt considerable trepidation in entering on the subject because Dr. Russell had warned him to be careful. He would have liked to hear about the epidemic form of the disease, because he knew of three epidemics which occurred recently, two in South Africa, and one at Stamford in England, the latter with fifty cases. He asked whether, in Dr. Russell's opinion, complete recovery ever took place in a case of acute anterior poliomyelitis, for he spoke very guardedly on that point. He also pointed out that physicians should prevent deformities taking place, but he did not even hint how that was to be done. He (Mr. Tubby) knew nothing better than a malleable iron splint, which should be bent to any desired angle. It was simple, inexpensive, and very useful. It was most important to arrive at an intelligent anticipation of deformities, and knowing which muscles were paralysed one could tell in what direction deformities would occur. Mr. Robert Jones and he, as shown in a book they had recently published, had come definitely to the conclusion that, so far as infantile paralysis was concerned, the outlook in any given case was very much better than could be anticipated at the time when the patient was first seen if appropriate treatment were adopted. They were also convinced that one of the great obstacles to recovery of a muscle partially or entirely paralysed was continuous stretching. That they had both been at considerable pains to emphasise by cases with full details. The first thing to do with a completely or partially paralysed muscle was to relax it, and therefore he was opposed to exercises against resistance as advised by Dr. Risien Russell. A paralysed muscle should not be stretched, but should be given time in which to recover. He would only allude in that connection to dropped wrist. If the extensor muscles were rested and massage carried out, recovery began to take place comparatively rapidly. If the muscles, however, were stretched indefinitely, progress would be very slow, notwithstanding massage. Mr. Jones and he found in in cases of infantile paralysis two factors. One was the effect of the paralysis itself, and the other the superadded deformities, which were the results of mal-position, of gravity and of contraction. Those two conditions had to be dissociated. It was necessary to work backwards, to remove the effects of the mal-position, and afterwards treat the paralysis. That seemed to him (Mr. Tubby) to be a most important distinction, and one on which great stress should be laid. Tenotomy was an unscientific operation in those cases, except under special circumstances, because it did not add to the power of the weaker muscles, but subtracted from the power of the stronger ones. It was accordingly necessary to devise other means. These were three in number: tendon or muscle transplantation, arthrodesis, and nerve grafting. Much had been written on the subject of tendon transplantation and the subject was gradually settling down. He agreed with Dr. Russell that the time to do tendon transplantation was when medical methods could do no more. What that date might be depended on the assiduity with which they had been carried out, and on the extent of the paralysis. In undertaking tendon transplantation, the central fact was that the surgeon must make up his mind exactly what it was he intended to accomplish by it. And in order to do that it was necessary to arrive at a determination of the main functional use of the part upon which the operation was to be performed. In the case of the leg, its main function was to support the body weight, and the finer movements were secondary in importance. Therefore tendon transplantation, other things being equal, should not be undertaken in a leg until it was clear that the support was the best which could be obtained. The great value of arthrodesis consisted in fixing a flail-like joint. In the upper limb the conditions were reversed; support was a secondary matter, the finer movements being of prime importance. In one case, at the instance of Sir William Gowers, he made an effort to obtain movements of interossei muscles by grafting off a portion of the extensor tendons on to the interossei muscles. The case turned out to be partially successful. With regard to nerve grafting, it was difficult to contrast it with

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tendon grafting, but he strongly agreed with Dr. Beevor that nerve transplantation should be rather reserved for cases in which not all the muscles were paralysed, and in which not all the irritability of the muscle had disappeared. Inasmuch as nerve grafting went to the very root of the matter and had proved successful in a certain number of cases, and inasmuch as when that failed one could still perform tendon grafting with satisfactory results, he looked forward to the future of nerve grafting with very great hope. In a case of paralysis of the deltoid muscle due to anterior poliomyelitis which he essayed to nerve-graft, he attacked the fifth cervical nerve, and the problem was as to where in the cord the fibres of the deltoid ran, whether on the inner side or the outer side. He thought the fibres were situated on the outer part of the nerve-cord. With the help of his colleague, Mr. Roch Carling, a strip was raised from the outer part of that cord, and grafted into the sixth nerve. He saw the patient at intervals, and fourteen months afterwards the deltoid had increased in volume, and the arm was showing partial power of abduction. It was necessary to have more research carried out in regard to nerve grafting, so that one might know exactly where in nerve trunks to look for certain fibres. Arthrodesis was of the greatest value in fixing the proximal joint in the upper limb, as, for example, the shoulder, when the muscles below were intact and capable of acting on a limb in which the shoulder muscles were entirely paralysed. If the shoulder were fixed by arthrodesis, the muscles below the elbow acquired a fixed point to work from.

Dr. LEONARD GUTHRIE said he, like Dr. Beevor, had little to criticise in the introduction. He had been glad to hear Dr. Russell refer to the value of electrical reactions in the acute stages, and he thought the opener's general idea was that the electrical reactions were not of such value as they were usually held to be. He (Dr. Guthrie) would go further and say that those reactions were to a great extent useless in the early stages, for even if there were absence to faradism and galvanism, it did not follow that the reactions would not return later on. He also maintained they were useless because it was possible to discover all that was needed about a paralysed limb without the use of the reactions at all. And more important than those was the reason that it was a very painful and tedious process to a child to take the electrical reactions, particularly in the acute stage. It had frequently happened to him that a child was admitted to hospital who had never previously had any electrical applications, and some enterprising and enthusiastic house-physician at once proceeded to take the electrical reactions, with the result that the child, which was perfectly docile before, was so terrified at the doctor that it might be a long time before it would submit to examination of any kind. He agreed that the reactions should be taken later, but not until the child was used to the sensation of electricity. His opinion was that the prognosis was better in the supposed afebrile cases, if there were really such But he thought there was constitutional disturbance in all, and that it was only a matter of difference of degree. In afebrile cases, one was commonly told that the child had nothing the matter with it until one morning it woke up with a paralysed limb. But on further investigation it was found that there had been some recent febrile condition, which was attributed to influenza or to over-eating. With regard to the treatment, Dr. Russell had excluded pathology from the scope of the discussion, and he (Dr. Guthrie) did not see how much progress was to be made in regard to treatment unless the cause were inquired into and considered. If it were an infectious disorder, it was desirable to attempt to find what the organism might be which caused it, and invent an appropriate vaccine. But he would not hold out hope that the treatment would be any the better for it, because by the time the diagnosis of infantile paralysis had been made the mischief would have been done. He did not believe that counter-irritation was of great value in the disease. Leeches might be, and dry-cupping was sometimes useful. With regard to posture, as the disease affected the ventral horns it seemed better to put the child on the back than on the face. In the treatment of the paralytic condition he agreed as to the immense value of massage, and that it should be skilled; it should not be left to any rubber or shampooer, or to the mother of the patient, or to an uneducated nurse. And it should not be undertaken until there was no pain produced. He attached far more importance to the

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re-education of the movements than was generally attached to that. One often saw a child which was supposed to be paralysed in both lower limbs, because it had never been taught or encouraged to use such power as it possessed. It was of the utmost importance to teach such a child to make use of the power it had. Next to massage, he regarded re-education of the movements as the most important point in the treatment of such cases. Besides the maintenance of nutrition of the muscles and the increased circulation induced by those means, there might be another reason for their employment. Mott had shown that the neurons were stimulated by stimuli from the periphery, which were supplied by ordinary movements of joints and muscles. In the paralysed limbs there were no such stimuli, and massage and movements and electricity tended to supply the deficiency. He believed faradism was of more use than galvanism, and came to that conclusion many years ago when a nurse told him she noticed that when he left off giving galvanism the patients improved far more by the use of faradism only. The local application of galvanism often caused pain, and it was impossible to inflict pain upon a paralysed child. As Dr. Russell had suggested, it was generally better to supply the current by means of the child's bath. It was never necessary to use a current which was painful. Dr. Russell had said he did not think strychnia would do any harm if it did not do good. But he (Dr. Guthrie) thought it would do harm if given in the old way by gradually increasing doses. He did not believe in ergot for the affection at all. He had been very much interested to hear Mr. Tubby's most useful remarks on the subject of the surgery of the condition. His own experience had not been very great, but he had sometimes thought that the improvement imputed to surgery was often due to careful after-treatment. Cases of the condition were naturally very tedious and prolonged, and even nurses might lose heart over them, but when surgery was called in there was a wakening of interest and everybody combined to make it a success. And if the parents had paid a heavy fee for the operation every effort was made to ensure that it was not wasted.

Dr. F. E. Batten said he had tried on one or two occasions to put a child suffering from the condition into the prone position, but he had convinced himself of the futility and discomfort of such a course, and he did not see any advantage from it. The application of galvanism had been rather strongly advocated by Dr. Russell, but his own experience did not agree with that. Children were very susceptible indeed to galvanism, and if it was applied in a bath the strength of current which could be so applied without causing undue pain was extremely small, so small that he did not think it would have any effect on the muscles. He agreed that it was of value as a skin stimulant, and a weak galvanic current might be of service where there was much vaso-motor disturbance. But usually its application was not worth any small benefit which might accrue. He agreed with Dr. Russell's remarks about the value and importance of skilled masseurs for the condition, but he would like to know what Dr. Russell did in regard to the out-patients who could not afford to come to the hospital regularly for skilled massage. He (Dr. Batten) was in the habit of seeing many out-patients, and practically it became a matter of educating the mother to use massage to the best of her ability. If parents were properly instructed in the matter very much good would accrue to the child. As to whether acute anterior poliomyelitis ever completely recovered, he had seen only one case in his own experience in which that undoubtedly occurred. If it were going to be recovered from he thought that took place quickly. Possibly in such cases the diagnosis might legitimately be called into question, but in his own instance others were also of the opinion that it was a case of the disease, and there was complete recovery.

Mr. Robert Jones (Liverpool) congratulated Dr. Russell on a most admirable paper, which was beautifully expressed. His own views in regard to the surgery of acute poliomyelitis were so much in accord with those which had been stated by Mr. Tubby that he had not much to add. He would, however, emphasise certain fundamental principles, more especially as they had much to do with surgical treatment, and largely affected the success or failure in any given case. He would refer especially to the stretching of tendons. Physicians and surgeons alike must realise that a strong muscle, if stretched sufficiently severely and long,

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will lose the power of action, and also that an overstretched muscle will regain its power if relieved from strain. If this important principle be lost sight of the surgeon will fail to discriminate between a muscle paralysed because of destruction of a motor cell, and a muscle not acting because it happens to be overstretched. As an instance he would take a case of old anterior poliomyelitis affecting the extensors of the hand, such as one might see going about the streets with his hand curled up, apparently unable to make any use of it. Yet notwithstanding that the paralysis may have lasted very many years, if appropriate treatment were carried out a very remarkable recovery may occur. This was due, not to any influence produced upon the nerve cells, which had already recovered to their full, but to the adjustment of the mechanical disability which had resulted from want of balance between muscular groups, which, unless treated, might persist for life. In answer to Dr. Russell's question, How long should a case be treated before deciding that the muscle would not recover? that depended very much on the treatment to which that muscle has been subjected. In the case, for instance, of the long-standing poliomyelitis of the hand it was in the power of any surgeon or physician, with no expert knowledge except the ability to distinguish a paralysed from an overstretched muscle, to say to that patient that in twelve or eighteen months his hand would recover its movement. The test was a simple one. With the hand held flexed by the patient, the tendons were further stretched by the surgeon and made more tense, and if the patient were able to extend his fingers even a little from that position there were present the potentialities of recovery, and it only remained for the muscles to be placed into position of rest and relief from tension. The treatment consisted in putting the hand on to a splint in a position of hyper-extension until the restoration of balance between the extensors and flexors was complete. This might, in a case of many years' standing, occupy even twelve months. This was a clear instance of the practical effect of recognising the difference between a paralysed and overstretched muscular group, between a postural and an organic lesion. So important did he believe this principle to be in the surgery of poliomyelitis that he would again illustrate its application by two of many cases which he had encountered. In paralysis of the shoulder girdle and upper arm, where the member lay limp and useless at the side, the hand alone functioning, it was his habit to remove a flap of skin from the forearm and the upper arm, and to fix in this way the arm in acute flexion, so that the function of the hand could be utilised for many useful purposes about the face and neck. In a girl, æt. 13 years, who had been affected for years in this way, the electric reactions denoted complete paralysis of biceps and brachialis anticus, and he therefore performed this operation. Many months afterwards, the skin being slightly stretched, the angle at the elbow was not so acute, and, to his delight, the girl was able to flex her elbow a few degrees. In fact, restoration of function had taken place in the paralysed brachialis anticus and biceps by relieving them from strain and allowing structural shortening to take place. Another case similarly paralysed, of seven years' duration, was most instructive. During those years a flicker of contractility had been observed in the biceps, so slight as to have no effect on the forearm, which lay limp and extended. Mr. Jones slung the arm and fixed it in acute flexion by seal and bandage, so that its position could not be disturbed for some months. Power was soon manifested both in the brachialis anticus and biceps, and by degrees the arm was lowered to right angles. From this position the patient could voluntarily acutely flex his arm. The patient's doctor, thinking the arm was well, removed the sling. The sequel was interesting: the weight of the arm so stretched the recovering muscles as to again render them useless, and function was regained only by further flexion aided by skilled massage. The bearing of this last case on tendon transplantation is obvious. It shows we should not expect a transplanted tendon to act if it is subjected to strain, and it shows the fallacies of expecting a newly-transplanted tendon to thrive against the combined forces of opposing muscles and the erratic deflection of body weight. A transplanted tendon required most skilfully-directed care. Mr. Jones preferred to confine his observations to the evils resulting from the overstretching of muscles, because it might be of value to physicians desirous of lessening the tasks of surgeons, and the principles involved were fundamental in their application to nerve anastomosis, tendon transplantation, and arthrodesis.

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Dr. FARQUHAR BUZZARD thought the whole field of the subject had been adequately covered in the discussion except prophylaxis, briefly referred to by Dr. Guthrie. That feature of the subject deserved further consideration. He regarded acute poliomyelitis as one of the most terrible diseases which had to be dealt with. In many respects it could be put almost on a par with smallpox: in the one physical disablement was almost inevitable as a result, and in the other facial disfigurement. At present it was desirable to know very much more about the occurrence of acute poliomyelitis, and about its frequency, and if inquiries were made he thought it would be found to be a much more common disease than it was usually supposed to be. There was great need here for a State department of health, for which the profession agitated. From such a source there would be information available about its frequency, and how many cases occurred every year, especially in a place like London? In some way it might be possible for those whose special knowledge enabled them to make investigation to see the cases in their early stage, with the result that accurate knowledge might be obtained which might lead to its prevention. There was reason to hope from the immunity conferred by a single attack of the disease that some vaccine might be discovered which could be used beforehand. This would be infinitely better than the limited means of treatment after the disease had established itself. Epidemics of the condition were now being more recognised, even if they were not actually more frequent, and there was strong ground for notification of the condition. Anything that could be done after the disease was established was as nothing compared to the blessings which would be conferred on mankind by a successful prophylactic measure.

Dr. F. J. POYNTON asked Dr. Russell if he could say what governed the arrest of the growth of bones in cases: it occurred more in some than in others. As a physician he would endorse what Mr. Tubby and Mr. Robert Jones had said. He had been much struck by seeing cases coming to hospital walking on a disabled leg in a faulty position, and in that way stretching their muscles and tendons and doing themselves very much harm. And for some years now he had been working hand-in-hand with the surgeon to check that complication. He had also observed a class of case in which the muscles, particularly those below the knee, were involved, and in which there was no history of acute poliomyelitis or of any illness. The wasting sometimes had only been recently noticed, and not the least clue was obtainable as to its origin. He did not confuse these with myopathies. Probably it was a chronic form of poliomyelitis. Poliomyelitis seldom ended in death, and as regards prognosis he had noticed that cases in which there were certain isolated paralyses of groups of muscles, rather than those of the entire limb, that these were more difficult to manage than the latter, for these were complicated by secondary deformities. In connection with polio-encephalitis inferior it was interesting to see how sudden the onset might be, and that it might be associated with a fall and injury due to the abrupt commencement, the child being taken suddenly ill. Occasionally such cases might for that reason be mistaken for fractures of the base, for meningitis, etc., and in that way a very faulty prognosis was possible.

The PRESIDENT (Dr. J. K. FOWLER) said he thought all must have regretted that pathology was omitted from the scope of the discussion; he believed much of interest would have been heard in that respect. What had been discussed was obviously not the treatment of the disease at all, but the treatment of the effects of the disease. Medical men would never get beyond that stage until they could treat the disease itself. He had been glad to hear Dr. Russell insisted upon massage of a skilled kind. Massage was a method which started in this country under favourable auspices, and remained in it for a considerable time. Now it had been fortunately revived, and he hoped some day there would be a massage department attached to every hospital where nurses and students could learn how to do it properly. He was also pleased to hear the expressions of opinion as to the length of time after which such cases could improve. All must have seen cases in which, owing mainly to the persistence of the mother's idea that improvement would occur, improvement had set in after a long time. He asked whether Dr. Russell was aware of any statistics showing the duration of life of such cases, *i. e.*

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whether, when recovery had occurred, life was shortened. That was a point which had some bearing on life assurance work.

Dr. RISIEN RUSSELL, in reply, thanked the various speakers for their participation in the discussion. He was not aware of special statistics on the subject, but the general opinion seemed to be that, except in cases where the muscles of respiration where involved, and where progressive muscular atrophy was superadded, the duration of life was not materially shortened in subjects of the disease. All the cases in which progressive muscular atrophy had been superadded were like the case mentioned by Dr. Beevor, in which the leg was the limb involved. He agreed that where skilled massage could not be obtained the next best would have to do, but wherever possible patients should attend at hospital to have massage done skilfully, particularly at the special hospitals where the patient did not live too far away. He regretted that there had been no definite pronouncement by the meeting on the question of the difference of prognosis according to whether the disease affected children or adults. Dr. Beevor seemed to doubt whether it was more severe in adults than in children. The point he wished to make was that poliomyelitis was very common in children, and in children all degrees of the affection were met with. But in his own experience all the adult cases had been very severe and the residual effects had been great. With regard to the possibility of complete recovery from the affection, Dr. Batten was able to find only one case in his experience, and he (Dr. Russell) had not seen a case of complete recovery, though improvement had been very great in many. In some cases the only disability remaining was in connection with the thenar eminence of one hand. And there was, in some cases, retardation of development of bones, but why that retardation should be greater in some cases than in others he could not say. He had hoped to hear something definite in that respect from Mr. Tubby and Mr. Robert Jones in regard to arthrodesis, *i. e.* whether and when one was justified from that point of view in interfering with the development of the limb—in other words, if it was the best method, how soon or how late should it be done? All must have felt grateful to Mr. Tubby and Mr. Jones for their lucid statements about the surgery of the condition, and he had learnt a great deal from what had been said. He bore Mr. Tubby's criticism that he (Dr. Russell) had not suggested any means whereby deformities were to be avoided: he purposely omitted that in order that the surgeons might tell them, and that had been generously done. It had been generally stated by authorities that it was in febrile cases with marked reaction which were the more favourable in prognosis. But Dr. Guthrie had rather surprised him in the view he took about faradism. His own experience was that it was of no use to employ a current in those cases which did not produce a reaction of muscle. If faradism produced contractions, well and good. But if the faradic current did not produce contraction, it was useless to employ it in the treatment of the disease. On the other hand, a muscle would persist in its reponse to galvanism a long time after it had ceased to respond to faradism, and, as Dr. Beevor had said, no muscle should be given up as hopeless until it had failed to respond to both. As to Dr. Batten's point that enough current could not be got into the water to treat a case of infantile paralysis, he could only say that that was contrary to his own experience and to that of that high authority, Dr. Lewis Jones, who was the first person to teach him the value of that method of treating it in St. Bartholmew's Hospital, from which Dr. Batten originally came. A child would bear it of sufficient strength, given through the water, to cause contraction of muscles on reversing the current in a way which would be hopeless on trying to apply the current direct to the limb. It would be impossible to deny the importance of what the President and Dr. Farquhar Buzzard mentioned in regard to prophylaxis in the disease. He did not venture to introduce that aspect into the discussion, because he thought he might be justly accused of talking of something which they knew nothing about. He shared the hope that in the future information of the kind would be available which would enable the organism responsible for the disease to be isolated, and possibly might even arrive at a time when a vaccine would be found to prevent the occurrence of the disease. The scope of the discussion did not permit of the inclusion of what Mr. Tubby spoke about in regard to epidemics, nor did he think it well to include the subject of polio-encephalitis. And in spite of what the President had said, he honestly thought it best that the pathology was not included if members desired to reach home before midnight.

*June 1st*, 1908.

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# DIAGNOSIS AND TREATMENT OF FRACTURES OF THE LONG BONES.*


Mr. A. PEARCE-GOULD. Mr. W. G. SPENCER.
Mr. A. E. BARKER. Mr. ROBERT JONES.
Mr. W. ARBUTHNOT LANE. Mr. LYNN THOMAS.


Mr. A. PEARCE-GOULD, in opening a discussion on the diagnosis and treatment of fractures of long bones, stated that his intention was to suggest certain points on which professional opinion was not yet decided, with the hope that many surgeons would express their opinions on these points. The well-recognised and classical signs of fracture, suddenly produced deformity, localised pain, localised tenderness, and oftentimes mobility in the length of the bone, with crepitus, were sufficient to lead to the recognition of the great majority of fractures, and the introduction of other means, particularly skiagraphy, should not lessen the thoroughness of the investigation of these older symptoms of fracture. He emphasised the great value of anæsthesia both in arriving at a correct diagnosis, and in applying proper treatment to a recent fracture. By the use of X rays a fracture could be demonstrated, when by other means it would only be suspected, and in some cases where even the surgeon had not even previously suspected it. But in such cases it was possible to take the position that the recognition of the fracture had little or no bearing upon the prognosis of the case nor upon the treatment. He referred, for instance, to fractures of the carpal bones, to incomplete fissures in long bones, fissures in the scapula, and simple fissured fractures of the vault of the skull. He laid more stress upon the value of X rays in showing the exact condition of a fracture, and referred to the fact that even serious displacement in fractures of long bones might be concealed by the supervening

* A discussion at the Medical Society of London.

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swelling. He submitted that the importance of obtaining really accurate and complete knowledge of the details of a fracture by skiagraphy was one of the questions upon which professional opinion was not yet formed, and in this connection he alluded to the difficulty that often arises in interpreting skiagrams. The line of a fracture might be very indistinctly indicated and very easily overlooked. The normal epiphyseal junctions might be mistaken for fractures, and a little want of care in taking a skiagram may readily distort the shadow and present an appearance of displacement which did not really exist. In reference to treatment, he pleaded for a much more frequent use of anæsthesia to aid in the reduction of displacement, urged the importance of immediate reduction, and insisted that splints should never be employed for the reduction of a fracture, but only for the maintenance of the fragments in their corrected position. The important debatable point in treatment, however, he thought was connected with the necessity for the exact and perfect adjustment of fragments in all cases of fracture. Pointing out the peculiar construction of long bones, and the perfect adaptation of the arrangement of the bony lamellæ for the transmission and distribution of pressure, and resistance to the pull of muscles, the question arose whether a perfect restoration of function after a fracture of a bone could be expected

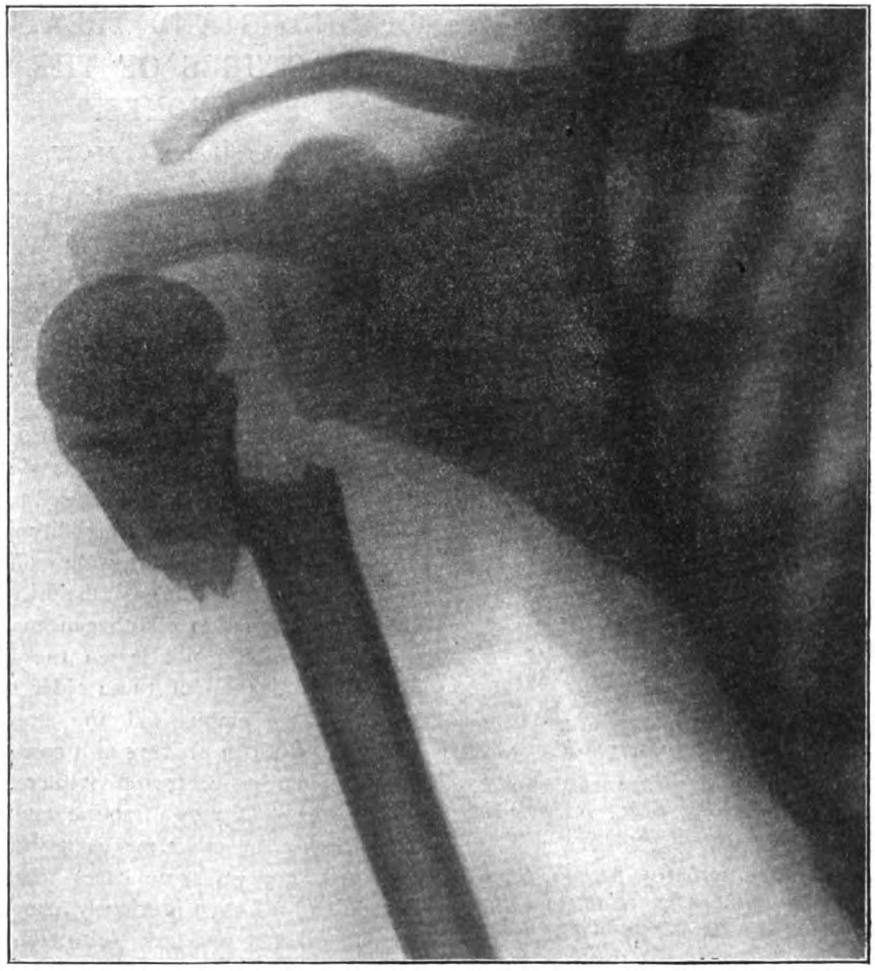

FIG. 1.—Before operation.

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unless the replacement of the fragments was so exact as not to cause any interruption in the mechanical adaptation of the bone to its special purpose in the skeleton. It was not easy to give a definite answer to this question, for while a study of the mechanics of the skeleton suggested that the structure of the bones was most closely connected with their function, experience of fractures showed that even considerable alterations in the shape of a bone might, under some circumstances, not be very harmful. He referred for instance to the results obtained in fracture of the middle of the clavicle and in Colles's fracture. In the more important weight-bearing bones of the lower limb he held that serious deformity of the bone was attended with distinct functional imperfection. Upon the answer given to the question depended the real value of the operative treatment of simple fractures. He thought it should be stated that these operations were not easy, that they were attended with rather more than the ordinary risks of modern surgical operations, that in some cases the mechanical difficulties were very great indeed, and that the use of screws, wires, staples and plates, while undoubtedly of value, was also attended with disadvantage, for they added to the local injury, and lessened the rapidity with which the final repair of the bone was effected. He therefore suggested that when employed, the procedure should be of the simplest

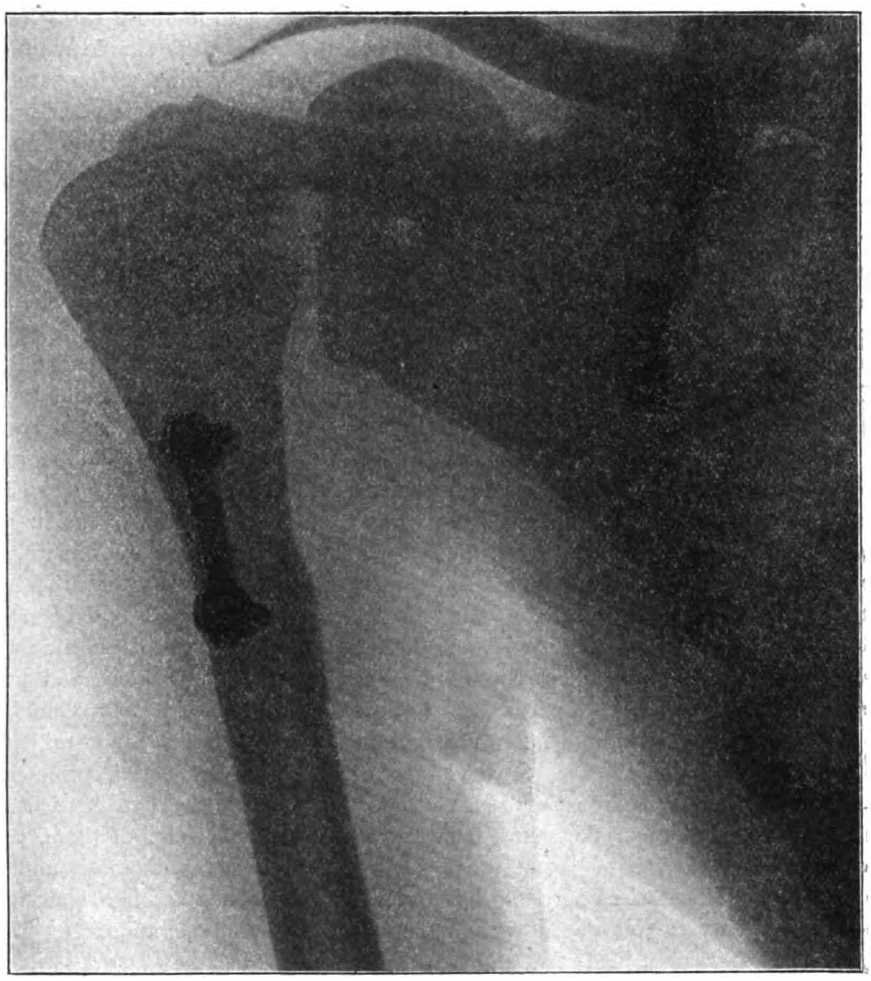

FIG. 2.—After operation.

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kind, provided that it was also efficient for its purpose. For example, a wire ligature surrounding fragments would often hold them in place, and if so was preferable to anything introduced into the bone. He referred to the value of massage, and in reference to movements insisted on the much greater usefulness of active movements than of passive movements, and spoke of the great improvement that could be obtained in fractures near and into joints by patient and gentle manipulations, and use of the part, when violent movements and rough handling only perpetuated the mischief. He thought there was evidence of a power of compensation for deformity caused by fractures, for some patients who at first were lame after a time lost all lameness and disability. It was an important point to ascertain how far this compensation for deformity could go.

Mr. A. E. Barker said that Mr. Gould had managed to invest a dry subject with a good deal of interest. On every point which that gentleman had raised he (Mr. Barker) was in agreement with him. The chief value of the address would be to direct the attention of the younger members of the profession to the possibility of doing without operation for fractures, and calling attention to the other means of diagnosing fractures accurately, without having recourse to X rays. There seemed to be growing up an idea that fractures could not be diagnosed without X rays, and that without the radiographer the surgeon was helpless. He protested against that. He had had some most elaborate diagnoses sent to him based upon skiagrams, but which were so obviously wrong that he sent them away. He did not think it wise to teach that every diagnosis of a fracture was dependent on radiograms. Besides, it was not likely that radiographers could be sent to different parts of the country, or that the country practitioner could have the necessary materials sent into the country. Moreover, this idea encouraged many surgeons to assume that perfect apposition and preservation of the contour of the bones was absolutely necessary, and that if that was not obtained one must resort to operation, and it prevented men who had a genius for mechanics from exercising it in providing a proper splint. Men now thought it was of no use devising a splint for a limb, but that was wholly a mistake. But here were cases in which it was very difficult to get anything like accurate apposition, and in those cases it might be justifiable to operate on the bone. He had not himself done many operations for recent fractures. Another exception was where there were compound fractures with great displacement. In many cases there was a wound which it was necessary to wash out and clean, and in those cases it might be well to wire the bone in the simple manner mentioned by Mr. Gould. Wiring of the patella stood on a different footing; there was a force present in that case always separating the bones, and therefore it was necessary to wire. He wired almost every case of patella fracture, and he had had no reason to regret it. All recognised the great value of massage, and one had reason to regret that intelligent massage was not always at hand. The best massage was required to produce the best effects. He thought there had been a tendency to trust too much to passive movement and to neglect active movement, and active movement should be commenced earlier, and it should be more vigorous. Many must have seen cases of stiff foot or stiff wrist from the hesitation of the surgeon who had treated it to use active movement. It was well known in military surgery that where men had had fracture about the ankle and been compelled to march, they had done better than men who had been put into an ambulance and treated for the fracture. He hoped Mr. Gould's thoughtful address would stimulate not only those present but the larger following in the profession to increase their study of the mechanics of fracture and the mechanics of reposition of them, and encourage everyone to make his diagnosis and do his treatment as much as possible without dependence upon X rays and operation.

Mr. W. Arbuthnot Lane said he acquiesced with a good deal of hesitation in the President's invitation to speak. That was due to the fact that his views, which he had expounded over and over again since he reported his first cases in 1894 at the Clinical Society, were absolutely opposed to those of the distinguished gentlemen who had spoken. He differed from them utterly. He thought it was the business of the surgeon to do his work as well as possible, in the treatment of fractures as well as in the treatment of any other

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part of the human body. Whether they thought so or not, public opinion and the X rays would compel them to do so, and the Law Courts had also to be reckoned with. He did not propose to weary the meeting by a repetition of his views, but he would produce some facts for their consideration. He had brought skiagrams of a few cases of recent fractures, the large majority being in young children, for the reason that he had previously shown so many in

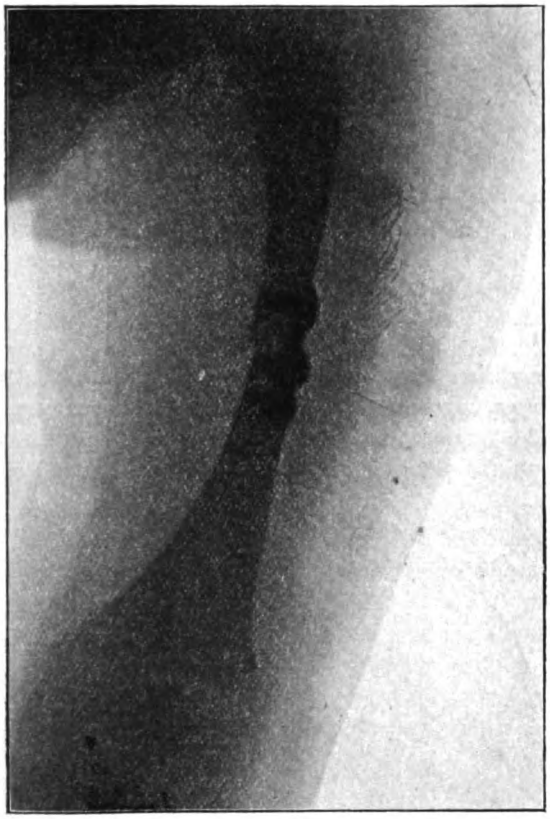

FIG. 3.—Result of operative treatment of a fractured femur.

adults. He also showed the skiagrams of a large number of malunited fractures on which he had had to perform extensive operations in order to restore the malunited bones to their normal shape and function. They represented only a few of the many he had operated on. They had previously been treated unsuccessfully by manipulation and splints, and he left surgeons to judge of the results for themselves. He thought that even the meanest intelligence would allow that the more perfectly

FIG. 4.—Before operation.

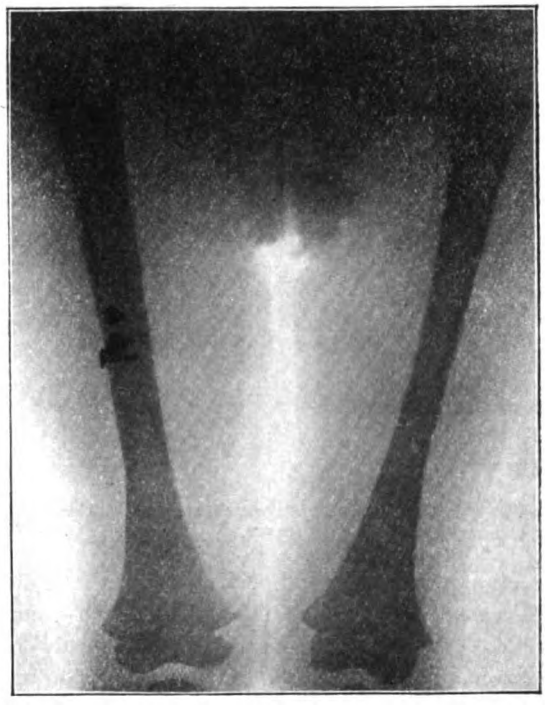

FIG. 5.—After operation.

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the skeleton was restored to its normal form the more perfectly was it likely to do its work; and the less perfectly it was restored the less perfectly would it do its work. Mr. Lane then handed round his photographs.

The first skiagram he showed was that of a

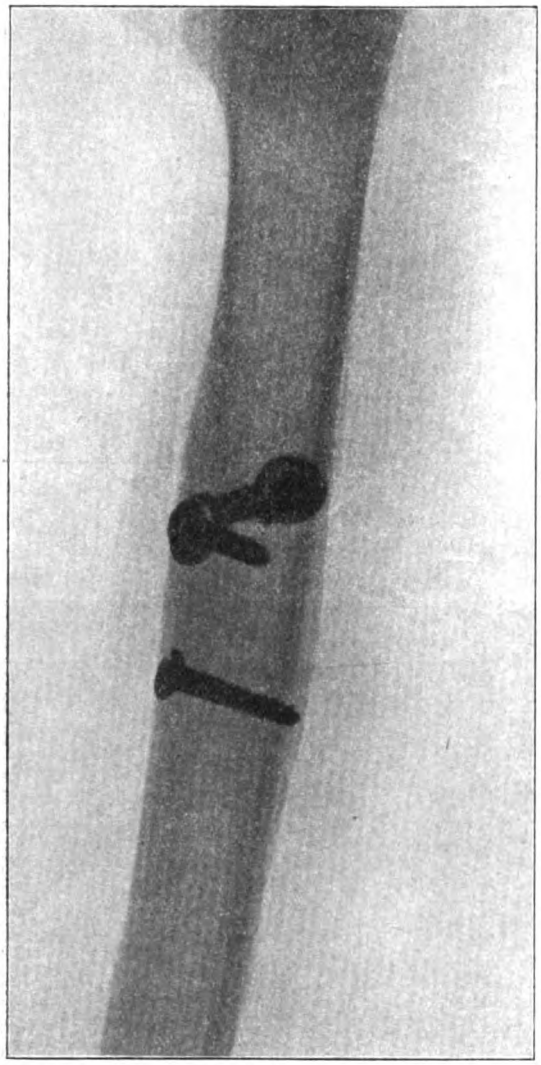

FIG. 6.—Result of operative treatment of a fractured femur.

fracture of the lower end of the humerus with displacement backwards of the epiphysis in a young child. It could not be reduced perfectly by flexion and manipulation. A vertical incision was made posteriorly, and by means of an elevator introduced between the shaft and lower fragment the bone was restored to its normal form and function.

The second X-ray photograph exhibited a similar

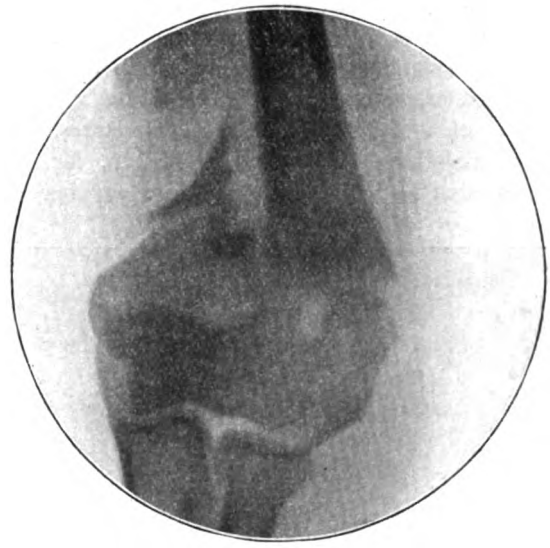

FIG. 7.—Before operation.

condition, except that the displacement of the lower fragment was inwards as well as backwards. The fragment was restored to perfect apposition and function by the same operative procedure,

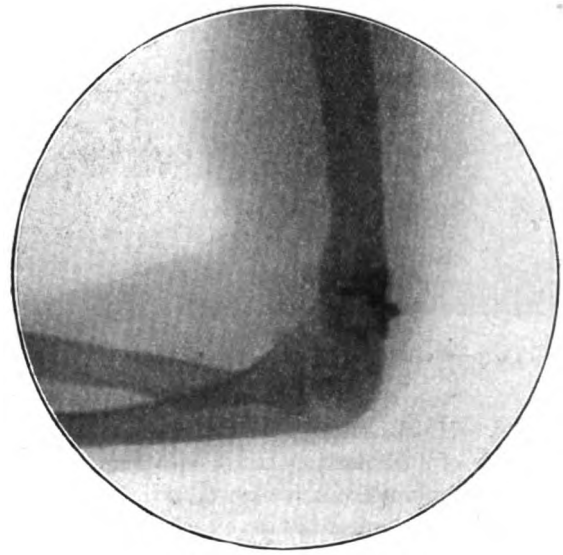

FIG. 8.—After operation.

namely, the fragment was prised into position by means of an elevator introduced through a posterior incision. In those two cases, as in almost all fractures about the epiphysis of children, it was

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unnecessary to employ wires, staples, or other means to retain the fragments in apposition when the bone had been restored perfectly to its normal form.

The third photograph showed a fracture through the upper part of the humerus in a child, æt. 7 years. The condition before operation was clearly shown in Fig. 1. All attempts at reducing this fracture being unsuccessful operative treatment was adopted four days after the receipt of the injury. Perfect apposition was effected and rendered permanent by plates and screws, the result being an anatomically and functionally normal limb. This satisfactory termination was shown in Fig. 2. During the operation it was clearly demonstrated that this result could not have been obtained by splints and manipulative procedures because of the mobility of the upper fragment, which difficulty was intensified by the small size of the upper fragment.

The next case was that of a fractured femur in a child, æt. 1½ years, which came under observation eight days after receipt of the injury. The fracture showed but little displacement, and several who were present thought it was unnecessary to operate, believing that traction would bring it into position. He exposed the fractured fragments which traction alone failed to replace. An elevator and forceps were used, and the bone was restored to its normal form and the fragments kept in accurate apposition by means of a plate and three screws. The result, which was anatomically and functionally perfect, was shown in Fig. 3.

The next case was that of a child, æt. 2 years and 6 months, who had sustained a fracture of the femur. She was sent to the Hospital for Sick Children to convalesce. She had been treated in a large general hospital in London, where the fracture had been manipulated under an anæsthetic and a plaster-of-Paris casing applied. Mr. Tyrrell Gray, the medical superintendent of the Hospital for Sick Children, finding that there was about three inches shortening, took the X-ray photograph of it shown in Fig. 4. It represented a long spiral fracture of the shaft of the femur. The amount of separation of fragments which was always present in such spiral fractures, and was directly proportional to the degree of overlapping, was also indicated in the illustration. The fragments were exposed by a long external incision and were brought into accurate apposition, being securely retained by means of a plate, screws, and silver wire (see Fig. 5). Abnormal difficulty was experienced because of the interval of time which had elapsed since the receipt of the fracture.

The next was another case of ordinary spiral fracture of the femur in an infant which came under observation seven days after fracture. One photograph showed the original condition, and the other after the fragments had been screwed in apposition by means of a plate and screws. This result of operative treatment was shown in Fig. 6.

Fig. 7 showed a T-shaped fracture with much comminution of the elbow-joint. It was treated by a triangular plate and three screws. The patient, a nurse at Guy's, now had a good working arm and could do her work. The condition after operation was illustrated by Fig. 8.

The next two photographs illustrated the consequences of the treatment of a precisely similar fracture by manipulation, splints, and massage, the result being complete ankylosis of the elbow-joint with associated disability. The contrast between the results of splint and massage treatment as compared with the results of operative treatment was demonstrated by these two cases.

The next photographs were from a case of a man who was thrown from his horse and sustained a bad fracture of the lower end of the tibia and fibula with considerable displacement of the foot. In this case the result of immediate treatment by operation seemed so excellent that the patient often forgot which leg was broken.

(*To be concluded.*)

*June 8th*, 1908.

WHEN serum from an individual with secondary syphilis is poured on top of serum from an individual in the later stages of syphilis, a ring forms at the line of contact, as in Heller's ring test. This ring is not observed with serum from persons free from syphilis. Similar tests with serum from persons with scarlet fever in its incipiency and late into convalescence or later, and with serum from measles patients during and after the disease, have given similar findings.—*Journ. A. M. A.*, vol. l, No. 21.

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# THE PATHOLOGY AND TREATMENT OF HEADACHE.*

By A. E. RUSSELL, M.D., F.R.C.P.
Physician to Out-patients, St. Thomas's Hospital.

To attempt to give a detailed classification and account of the various forms of headache would be impossible in the time at my disposal. Attention will therefore be directed to a few types, and an attempt will be made to ascertain whether any general rules may be formulated as to the pathological conditions underlying headache, and as to methods of treatment to be adopted. I do not propose to dwell on the many local affections of bones, sinuses, etc., which are prone to be associated with cranial pain, for in such conditions local symptoms of swelling, discharge, etc., are frequently present, and clearly indicate the direction in which investigation must be pursued with the object of forming a diagnosis.

It is rather with regard to intra-cranial pain that I wish to make a few remarks, and in order to focus our views it will be well to commence with a short consideration of the tissues which are responsible for the presence of headache. All the evidence at our disposal goes to show that the brain tissue itself is devoid of sensation. The dura mater, however, with the tentorium and falx, contain nerve-fibres derived chiefly from the ophthalmic and superior maxillary divisions of the fifth nerve, and it is highly probable that headache is due to afferent impressions from these fibres. An interesting observation bearing strongly on this point was made by Harvey Cushing, who noted that after excision of the Gasserian ganglion for severe trigeminal neuralgia, any subsequent headache was only experienced on the side with the intact fifth nerve.

### *The Headache of Cerebral Disease.*

Gross intra-cranial disease is nearly always associated with the presence of headache. Cerebral tumour is a well-defined local condition, and as it is slow in development any accompanying pain should be susceptible of study. Any growth at or near the cortex will readily give rise to pain from stretching of the dural fibres, and such pain may be an early symptom. A tumour situated more deeply in the white matter presumably need not of necessity cause pain in its very early stage. In the more fully developed stage of cerebral tumour, however, certain symptoms appear which are known as pressure symptoms, and under this heading may be included headache, optic neuritis, vomiting, coma and convulsions. These symptoms are found to be associated with, and are attributed to, an increase in the intra-cranial tension. Surgical intervention with removal of a considerable area of bone, and with resulting diminution of intra-cranial tension, affords either complete relief or marked amelioration of these distressing symptoms, so much so that the so-called decompressive operation is undertaken with this end in view, even when the evidence is conclusive that the nature and position of the growth are such as to preclude its removal. Not only this, but an operation at a distance from the site of growth may be as effective as one in its near neighbourhood. Such operation, however, is by no means free from risk. In tuberculous meningitis and other conditions associated with a rapid increase in the quantity of cerebro-spinal fluid headache is one of the most obvious and distressing symptoms. The attitude occasionally observed in an infant with tuberculous meningitis, with both hands held to its head, is an indication of the pain it experiences. The immediate, if only temporary relief of symptoms that occurs on the withdrawal of cerebro-spinal fluid by means of lumbar puncture in cases of tuberculous meningitis, cerebro spinal meningitis, etc., also demonstrates the grave effects produced by the presence of increased intra-cranial tension.

Measures such as trephining and lumbar puncture, capable of diminishing this pressure, relieve not only headache but other concomitant symptoms, such as optic neuritis, vomiting, coma and convulsions.

The effects of increased intra-cranial pressure are, therefore, severe and obvious. Experimentally, it has been found by Harvey Cushing that by increasing the intra-cranial tension in dogs up to a point equal to that of the general blood-pressure, *i. e.* to a point at which the blood-flow through the brain would be abolished, a conservative mechanism, viz. a rise in the general blood-pressure produced by vaso-constriction, is set in action. In other words, a rise in the intra-cranial pressure which

* The Annual Address delivered before the Southampton Medical Society.

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would be sufficient to impede the entrance of blood produces a conservative rise in the general blood-pressure by means of which the circulation through the brain is maintained. Clinically, we see evidences of this conservative rise in blood-pressure in the slow, high-pressure pulse observed in cerebral compression in some cases of cerebral hæmorrhage, tuberculous meningitis, etc. Cushing records an admirable example of this conservative process in a case of cerebral hæmorrhage.

The patient was a man, æt. 40 years, who was admitted to hospital thirty-six hours after onset of symptoms. He was in a state of profound stupor with right-sided flaccid paralysis. Temperature 99·6° F., respiration rate varied from 21 to 27, pulse 50 and of high tension, registering 300 mm. Hg. Conjugate deviation of eyes to right with nystagmus. Pupils equally contracted and reacted to light. Veins of forehead greatly distended.

*Operation.*—A large osteo-plastic flap was turned down over the left hemisphere. The dura was tense and without pulsation. On incising it the brain bulged far into the opening and began to pulsate actively. The convolutions were greatly flattened, deeply cyanosed, and the veins occupying the sulci were widely dilated, and their contents so dark that the colour contrast between veins and arteries was of an unusual degree. Blood was evacuated from the hemisphere by means of a grooved director, about two teaspoonfuls. *The blood-pressure began at once to fall, and in twenty minutes had reached the normal level.* The pulse-rate during this fall in blood-pressure remained unaltered. The release from intra-cranial tension was evidenced by the immediate collapse of the bulging hemisphere. On the following day the patient's condition had greatly improved. The pulse-rate and blood-pressure remained normal, respiration was no longer laboured, stupor was much less deep, and he could be roused with considerable ease. The conjugate deviation disappeared and he seemed to see and recognise objects. Stupor returned on the following day, though the blood-pressure only rose to about 160 mm. Hg.; œdema of the lungs set in and he died two days later. Post mortem a large hæmorrhage was found in the white centre of the left hemisphere.*

* "The Blood-pressure Reaction of Acute Cerebral Compression, illustrated by Cases of Intra-cranial Hæmorrhage," 'Amer. Journ. Med. Sci.,' June, 1903.

In this case we see a clinical illustration of the results obtained experimentally in animals. As a result of the effusion of blood into the substance of the brain the intra-cranial tension was increased, and the enormous increase in the blood-pressure (up to 300 mm. of mercury) was the conservative rise necessary to ensure the maintenance of the cerebral circulation. As soon as the intra-cranial pressure was lowered by trephining and evacuating the blood, the blood-pressure fell to normal. It is clear, therefore, that increased intra-cranial tension is a cause of headache and other severe symptoms, and that these may be markedly alleviated or cured by treatment capable of lowering such tension.

### *Headache in Nephritis and Uræmia.**

In cerebral uræmia we frequently see a symptom complex almost identical with that of cerebral compression, viz. headache, vomiting, drowsiness, coma, convulsions, optic neuritis, etc. This symptom-complex is, at any rate in great measure, dependent upon increased intra-cranial tension, for many cases are now on record in which extraordinary relief has followed lumbar puncture, a procedure which, by means of allowing some of the fluid to escape, diminishes the pressure within the cerebro-spinal space.

Thus Mm. Pierre Marie and Georges Guillain† record the case of a man, æt. 20 years, suffering from nephritis and epilepsy, who had suffered from severe headache for twelve months, with a high blood-pressure (200 to 210 mm. Hg.). Lumbar puncture was performed, the cerebro-spinal fluid being under greatly increased tension, as it spurted out from the needle; 6 c.cm. were withdrawn. Within five minutes the headache was materially improved, and within two hours it had completely disappeared. Relief was complete for two days, when the communication was read.

In the discussion which followed the paper M. le Gendre recorded a similar case of relief. A man, æt. 57 years, painter, suffered from arterio-sclerosis and plumbism with albuminuria. He was uræmic with headache, myosis and insomnia. Lumbar puncture was performed and 13 c.cm. with-

* The following extracts are from a paper by the author on "Uræmia," 'West London Medical Journal,' January, 1907.

† 'Bulletin et Memoires de la Soc. Med. des Hopitaux de Paris,' 1901, p. 427.

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drawn. The pressure was not noted. The same evening the headache was improving, and he slept well. The headache progressively improved, and in three days was absolutely gone.

G. Scherb* records a case of nephritis with uræmia in a man, æt. 49 years, suffering from very severe headache, and resisting all treatment. He was admitted to hospital almost comatose, with history of vomiting, auditory hallucinations and severe insomnia. Fundi showed slight venous stasis. Increased arterial tension. Incontinence of fæces. Lumbar puncture was performed and 20 c.cm. removed, the fluid issuing in a spurt under considerable pressure. Within five hours he was conscious and could answer questions. Next day he was quite free from headache and remained so for six weeks, when it returned. Œdema also appeared, limited to the region of the forehead, eyelids, cheeks, lips, ears and the skin of the scalp. Lumbar puncture was again performed, but with only temporary relief, and death occurred ten days later.

D. C. McVail records another very successful case: † A man with acute nephritis, with severe intra-cranial pain, followed by convulsive attacks repeated during thirty-six hours, became almost blind and finally comatose. He was œdematous. Lumbar puncture was performed, the fluid issuing in drops, and apparently under no tension. Within twenty-four hours there was a return of consciousness, and sight began to reappear. He slept well, and on the next day was free from pain, with perfect vision. This was followed by a rapid recovery from the renal condition.

Many other cases are on record, and it is clear, therefore, that headache in uræmia may be relieved by lumbar puncture with its attendant lowering of the cerebro-spinal pressure. That the headache is actually due to the increased intra-cranial tension is evidenced by the simultaneous relief of other concomitant symptoms of uræmia on lumbar puncture—convulsions and coma, for instance. Thus T. Arthur Helme‡ records a case of puerperal convulsions, in which two hours after child-birth a convulsion occurred with sudden complete amaurosis. The fits recurred during the day and became increasingly severe and frequent. In ten hours she had fifteen fits and was comatose. Lumbar puncture was performed, and a drachm and a half of fluid escaped under pressure. She had two further very slight fits; in a few hours she could speak, and sight rapidly and completely recovered.

F. Proud * records two cases of puerperal eclampsia in which the patients were moribund from convulsions and coma. In both there was a most rapid recovery after lumbar puncture, the fluid being evidently under pressure in one.

R. N. Willson † records three cases. Puncture was performed in two moribund cases of uræmia. In both the cerebral relief was evident. The respiration became much easier in both. The pulse dropped from 110 to 84 in one, and from 90 to 50 in the other. The third case was complicated by a large intra-cranial cyst.

In a further paper Willson brings his cases up to ten.‡ Of the ten cases six had convulsive movements, general in five and unilateral in one. *In every case the convulsions terminated on the withdrawal of the cerebro-spinal fluid*, and in only one did they return within several weeks of the operation. His best results were obtained in those cases in which the fluid spurted from the cannula, *i.e.* in which the pressure was greatest. In the autopsies of fatal cases (three out of the ten), increased cerebro-spinal fluid, dilated ventricles, and œdema of the brain were found.

F. C. Eve § notes the case of a man with uræmic convulsions associated with total blindness. The convulsions were relieved by venesection, but the coma remained. Lumbar puncture was performed (fluid under high pressure). Next morning

* "Confusion mentale et syndrome cerebelleux au cours de l'uremie; excellent effet de la lombo-ponction," 'Revue Neurologique' (de Paris); vol. x, 1902, p. 19.

† "Spinal Puncture in Uræmia," 'Brit. Med. Journ.,' October 24th, 1903, p. 1054.

‡ "A Suggestion for the Treatment of Puerperal Convulsions by Spinal Subarachnoid Puncture," 'Brit. Med. Journ.,' May 14th, 1904.

* "Lumbar Puncture in Puerperal Eclampsia," 'Brit. Med. Journ.,' March 24th, 1906, p. 678.

† "The Pathogenesis of Uræmia and Eclampsia," 'The Journal of the American Medical Association,' October 8th, 1904, p. 1019.

‡ "The Relief of Uræmic Hemiplegia and other Uræmic States by Lowering Intra-cranial Pressure," 'Transactions of College of Physicians of Philadelphia,' vol. xxvii, 1905, p. 34.

§ "A Cerebro-spinal Manometer," 'Lancet,' April 22nd, 1905, p. 1068.

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the man was quite conscious and had recovered much of his sight.

Headache, coma, and convulsions in uræmia are, therefore, seen to be at times relieved by lumbar puncture and the withdrawal of cerebro-spinal fluid. Such puncture frequently demonstrates the existence of an increased intra-cranial tension. The immediate relief of the symptoms on the withdrawal of fluid, with the necessary diminution of the intra-cranial pressure, compels the conclusion that the symptoms are due to the pressure.

We are not concerned here to discuss the cause of the increased tension, but it is possibly due to the cerebral œdema, which is so commonly found at autopsies on cases of uræmia.

In cases of chronic interstitial nephritis, in which headache is a frequent symptom over long periods of time, apart from any evidence of uræmia, the pain may be due to the increased blood-pressure so common in these cases, an increase in which the intra-cranial vessels are as much concerned as any others.

### *Headache in Epilepsy.*

Severe headache is frequently a prominent feature after an epileptic fit, and may last for several hours. Whatever hypothesis may be adopted to account for the production of epileptic fits, certain mechanical effects are of necessity produced as the result of the severe tonic and clonic spasms. With the onset of the muscular spasm the return of blood from the brain is materially hampered; indeed, it is probable that blood is actually forced upwards through the cervical veins into the brain. The valvular apparatus of the jugular veins is quite insufficient to prevent such reflux on the occurrence of severe muscular spasm. As a matter of fact the condition of the cerebral cortex during a fit was actually observed on one occasion by Sir Victor Horsley.* A fit occurred during an operation for cerebral tumour, and with the convulsive spasms the cerebral cortex became distinctly hyperæmic. Engorgement of the face and a sensation of fulness and of throbbing in the head are matters of common observation as the result of prolonged stooping.

We can, therefore, hardly be wrong in assuming that the intra-cranial tension is greatly increased during the convulsive stage of an epileptic fit. In the light of the preceding remarks on the results of increased intra-cranial tension it is not surprising that headache should follow. This rise of intra-cranial tension is also supported by clinical and pathological evidence. Thus Morgan Hodskins and Arthur Morton have reported a series of seven cases of status epilepticus treated by lumbar puncture.* In four out of the seven recovery ensued, and in every case but one there was an immediate and marked improvement as the result of the lumbar puncture. Further, in six out of the seven the cerebro-spinal fluid was evidently under increased pressure. Status epilepticus is, then, evidently associated with increased intra-cranial tension. It is not unreasonable to assume that the over-distended vessels would allow of undue transudation through their walls with a resulting cerebral œdema.

Finally, Mott† has shown that the essential changes in the brain in cases of status epilepticus are—

(1) Great venous congestion and stasis.

(2) Œdema of the brain and marked distension of the perivascular lymphatics; flattening of the convolutions and naked-eye increased vascularity; but "inasmuch as the brain is contained in a closed cavity, *venous stasis and œdema must be associated with a corresponding arterio-capillary anæmia.*"

The conclusion seems justified, therefore, that the headache of epilepsy is also the result of increased intra-cranial tension.

### *Anæmia and Headache.*

Headache is a common symptom in anæmia and especially in chlorosis. It is of interest to note that the hypothesis that this is due to an increase in the subarachnoid pressure has been brought forward by Lenhartz‡, and that Romberg§ has come to the conclusion that the optic neuritis

* "Origin and Seat of Epileptic Disturbance," 'Brit. Med. Journ.,' April 2nd, 1892.

* "Lumbar Puncture in Status Epilepticus," 'Boston Med. and Surg. Journ.,' 1905, vol. clii, p. 700.

† "Preliminary Communication upon the Changes in the Brain, Spinal Cord, and other Organs found in Persons dying after prolonged Epileptiform Convulsions," 'Archives of Neurology,' London, vol. i, p. 499.

‡ 'Münch. med. Woch.,' 1896, No. 809.

§ 'Berl. klin. Woch.,' 1897, No. 25.

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which does occur, though very rarely, in chlorosis is possibly due to increased intra-cranial pressure.

The pathology of chlorosis is a very difficult problem, but many of the symptoms of the disease are explicable on the view that there is a great increase in the total volume of the blood plasma. That this increase does exist receives strong support from the results obtained by the carbon monoxide method of determining the plasma volume, devised by Haldane and Lorraine Smith. Clinically, chlorotics are usually plump, and plumpness depends as much on the presence of an abundant plasma as on fat. The rapid wasting in children after a few days of diarrhœa and vomiting must be due in the main to loss of fluid from the tissues. The blood in chlorosis is very watery; it flows with the greatest ease on pricking the finger, and its specific gravity is much lower than in the case of normal blood. There is a tendency to œdema of feet and shins, a condition very readily brought about with an increased volume of watery blood, a factor which is also present in the case of nephritis with œdema. Bearing these points in mind, the hypothesis that the intra-cranial tension is increased owing to the vessels being overfilled with blood, and possibly by some œdema of the brain as well, appears, at any rate, feasible. It is not, however, one that is easily put to the direct test of lumbar puncture, as the condition yields so readily to ordinary treatment.

### *Migraine.*

The cause of the headache of migraine is a difficult problem. It has been ascribed to periodic discharges from the cortical centres, but such a statement carries little meaning. What is certain is that concurrently with the headache there are marked vaso-motor symptoms, and the theory that the attacks are vaso-motor in origin is worthy of consideration. It has been supposed that the earlier symptoms of the attack are due to vaso-constriction and the later ones to vaso-dilatation. The visual manifestations might well be due to vaso-constriction and the occasional occurrence of transient aphasia, hemiplegic weakness and tingling would seem susceptible of a similar explanation. There is an obvious disturbance of the peripheral vaso-motor regulation during an attack. Shivering is common and the radial pulse may be small and hard. The peripheral part of the temporal artery is sometimes firmly contracted and occasionally becomes arterio-sclerotic. On the other hand, the carotids throb, and the throbbing character of the headache suggests that the intra-cranial (and often the peri-cranial) arteries are over-full of blood. There is some direct evidence to show that this is so, for the vessels of the fundus oculi have been observed by Mollendorff* in an attack: "During the attacks the background of the eye on the suffering side was of a bright scarlet red, the optic papilla red and œdematous, the arteria and vena centralis retinæ enlarged, the latter knotty and very tortuous." The fact also that compression of the common carotid artery of the affected side will in many cases completely relieve the pain of migraine gives support to the view that the condition depends upon a vaso-dilatation of the intra-cranial vessels. As we have seen, the site of intra-cranial pain is in all probability the area of distribution of the trigeminal fibres in the dura and allied structures. The dura is closely adherent to the subjacent bone, and it is a very inextensile structure and a highly vascular one. Francis Hare suggests that the pain is produced by the vascular distension in these structures compressing the sensory nerves contained within them. The same argument applies, of course, to the peri-cranium and, in severe migraine and other headaches, pain radiating from the neck up the back of the head is often noticeably severe. Tenderness of the scalp is common. Compression of the back of the neck often affords the greatest relief, and the result is to be attributed to the pressure diminishing the flow through the occipital arteries.

I think, therefore, that much is to be said in favour of the view that migraine depends upon paroxysmal disturbances of the vaso-motor system, affecting especially the cerebral vessels. On what this depends is another question. General chilliness and even shivering is, as a rule, observed during the attack, and the face is usually pale. These conditions indicate a peripheral vaso-constriction. The whole vaso-motor regulatory mechanism is obviously at fault.

Up to quite recent times it was asserted that the cerebral vessels were devoid of nerve-fibres and that changes in their calibre were passive. It is now thoroughly established that a widespread net

* Liveing, 'Megrim and Sick Headache,' 1873, p. 307.

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of nerve-fibres is distributed to these vessels, and it has been demonstrated microscopically. Not only so, but evidence is beginning to accumulate from the experimental side that such nerve-fibres can be stimulated with demonstrable results.

Clinically, also, the evidence is fairly strong. Cases of recurrent amblyopia from attacks of narrowing of the retinal artery are on record.

The phenomena of Raynaud's disease also are best explained on the hypothesis of attacks of spasm of the peripheral vessels, and a remarkable case has been recorded by Osler* in which a woman who was the subject of Raynaud's disease had on four occasions transient hemiplegia, once left-sided and three times right-sided with aphasia, occurring with the attack of peripheral spasm. All these attacks were transient, and completely recovered from. It is impossible to believe, therefore, that any lesion such as hæmorrhage or thrombosis was present, and the only conclusion that is justified is that these cerebral attacks were also due to spasm of cerebral arteries.

I have made this digression with the view of showing that the intra-cranial vessels are susceptible of active changes in their calibre, and that, therefore, we need not hesitate to accept the vaso-motor origin of migraine on account of the old belief that these vessels are devoid of vaso-motor nerves. Another possible explanation of at least some cases of migraine will be considered at the end of the next section.

In connection with the above views on the causation of the headache in chlorosis, a paper by Dr. G. W. Ross under the title, "On the Relief of Certain Headaches by the Administration of one of the Salts of Calcium," is of considerable interest.† The headaches which he refers to present the following characteristics:

(1) Present and most severe on waking, and tends to disappear in from one to six hours.

(2) Usually a dull heavy ache or a frontal or temporal throbbing; less often occipital, vertical or unilateral.

(3) Very chronic, often of several years' duration, and most intractable.

(4) Associated with a deficient coagulability of the blood.

He further describes the expression as heavy and listless, face full, eyes often puffy; some anæmia is usual, or an actual chlorosis. Women are more commonly affected than men. Constipation is the rule; loss of appetite and indigestion common. There is a tendency to chilblains, urticaria and œdema, the latter manifesting itself more commonly as a morning fulness between the eyes and less frequently as an œdema of ankles or feet or legs. Such patients sleep heavily but wake without feeling rested, and there is a tendency to mental depression. Irritability combined with languor is characteristic.

Dr. Ross then refers to Professor A. E. Wright's researches into the pathology of chilblains, urticaria, certain œdemas and physiological albuminuria, which conditions Professor Wright considers to be due to a lowered coagulability of the blood in some way allowing the transudation of plasma from the blood-vessels into the lymph spaces—a process termed "serous hæmorrhage."

The association in a patient of severe chronic headache and of a troublesome urticaria along with deficient coagulability of the blood, and further, the vanishing of the headaches and urticaria on treatment with calcium chloride, suggested to Dr. Ross that the same "serous hæmorrhage" might be the basis of the headache. Dr. Ross treated forty-eight cases by administering 15 gr. of lactate (or chloride) of calcium three times daily. Forty of the cases obtained complete relief and eight considerable relief. Concomitantly with the relief the coagulability of the blood was exalted in all cases tested.

Chilblains occurred in six cases and urticaria in two. All these eight cases obtained complete relief of pain and swelling. In three of them the headache and chilblains reappeared on the decalcification of the blood by the administration of sodium citrate, to disappear a second time on treatment with calcium lactate.

In twenty-three cases there was pain after food, sixteen of which found complete and seven partial relief. Relief was obtained very rapidly. Dr. Ross attributes the pain to a "serous hæmorrhage in the wall of the stomach," and notes the frequency of gastric crises in urticaria. Osler also has dwelt on this in a paper on "The Visceral Manifestations of the Erythema Group of Skin Diseases."*

* "The Cerebral Complications of Raynaud's Disease," 'Amer. Journ. Med. Sci.,' vol. cii, 1896, p. 522.

† 'Lancet,' January 20th, 1906, p. 143.

* 'Amer. Journ. of Med. Sci.,' vol. cxxvii, 1904, p. 1.

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Œdema of the eyes or of the extremities occurred in twenty-one cases, and of these seventeen were completely and quickly relieved. Further, in two typical cases of migraine the paroxysms practically ceased during treatment with calcium lactate.

A most instructive case published by Osler* not only throws some light on the nature of at least some cases of migraine, but also materially supports the views upheld by Ross. The patient was a medical man, æt. 29 years. From childhood he had suffered from indigestion. At the age of twelve he had an attack of right hemiplegia and aphasia which lasted for a week or ten days. Within the year he had five or six further attacks of hemiplegia, each successive one less severe and not accompanied by aphasia, and from then onwards to the time of examination (1898) he occasionally felt a numb tingling sensation in the side. In 1893 he had a severe attack of lumbago, in 1895 influenza, and from then onwards became subject to severe attacks of migraine. In 1896 he had a mild attack of rheumatism. In February, 1897, a sudden attack of swelling and pain occurred in the feet, and at the same time he began to suffer from soreness at the end of fingers. He had frequent attacks of angio-neurotic œdema of the upper lip, and outbreaks of urticaria associated with severe darting pains in the legs. In May, 1897, he suffered from an attack of abdominal colic with pains in the calves of the legs and an outbreak of purpura and urticaria; in 1898 from hæmaturia and albuminuria. Professor Osler considers that the recurring attacks of hemiplegia were probably associated with changes in the brain of essentially the same nature as those which subsequently occurred in the lip and skin. In other words the assumption was that an angio-neurotic œdema occurred in the brain substance. Presumably the anæmia thereby produced was the factor which precipitated the attacks of hemiplegia. If this œdema of brain were not associated with any increase in the intra-cranial tension there is no reason why headache should have occurred. It is noteworthy, however, that the patient later became subject to attacks of migraine, and without wishing to stretch the comparison too far, the thought arises that the attacks of migraine may also have been due to an œdema of a portion of the brain, not of the motor region, but possibly of the dura mater, with resulting compression of nerve-fibres, or a more widely spreading œdema with increased intra-cranial tension.

* "The Visceral Lesions of the Erythema Group," 'Brit. Journ. Derm.,' vol. xii, July, 1900, p. 227.

*Hypermetropia.*

Hypermetropia with its attendant spasm of the ciliary muscle is another condition frequently associated with headache. The nature of this form of headache is not clear. There is presumably no alteration of the intra-cranial tension, and there is little evidence of any interference with the flow of blood through the brain. Possibly the spasm of the intra-ocular muscles gives rise to a reflex neuralgia of the sensory fibres of the cranial membranes. Such headaches yield readily on proper correction of the error of refraction.

If we try then to specify the main factors responsible for the production of headache, the first would be direct irritation of the sensitive membranes of the brain as by a cortical tumour, meningitis, whether acute, as in suppuration and tuberculous meningitis, or whether chronic, as in syphilitic disease.

The second would be an increase of the intra-cranial tension, which may occur together with or apart from the conditions just mentioned, or in their later stages.

The third would be intra-cranial vaso-motor change as seen especially in migraine. It is probable that many, if not the majority of headaches, other than those typically migrainous, are produced in much the same way. Most headaches are intensified by anything which tends either to raise the general blood-pressure, such as exertion, or by anything which tends to hinder the free exit of blood from the cranium, such as stooping. Most headaches, again, are accompanied by vaso-motor phenomena, such as chilliness or actual shivering. In rigors cutaneous vaso-constriction is a conspicuous feature, and headache is usual and often intense.

In typical migraine the recurrence of the attacks over years has given opportunities for minute study of the phenomena by the patient, and the condition has established itself as a clinical entity. But the isolated attack in a patient who has perhaps only two or three in a lifetime is simply a "headache" or a "bilious attack." The nausea and vomiting

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are apt, then, to be made the keystone of the arch. In the one case the tendency to recurrence of attacks constitutes migraine; in the other the few attacks occurring at long intervals are not recognised as coming under the same category. The term "headache," too, is misleading, inasmuch as it refers solely to the pain in the head without reference to the languor, anorexia, nausea, vaso-motor changes, etc., some of which are such characteristic accompaniments of the pain.

The vaso-motor system is a part of the great sympathetic system, and is not under our voluntary control. Designed as it is to respond to the varying demands of the organs and tissues of our body by regulation of the blood flow, it possesses two types of nerve-fibres, a motor or vaso-constrictor and an inhibitory or vaso-dilator, which are set in action by mechanical, thermal, chemical and other forms of stimulation. Whilst we are unable to exert any direct volitional influence over this system, it is clear that we can impose excessive demands upon them by such errors as excess of food, qualitative or quantitative. If the great alimentary tract is overloaded these fibres will be given undue work, and may, and probably must, suffer in efficiency as the result, either in the direction of fatigue or undue irritability. W. Russell * attributes great importance to the influence of excessive food products in the blood as direct excitants of contraction of peripheral arteries—hypertonus, and also to the retention of waste products in people leading sedentary lives, especially if associated with constipation. The influence of faulty food habits on the development of arterial disease has received recognition in late years. Francis Hare attributes the abnormal vaso-motor changes of migraine to excess of nutrient material in the blood (chiefly of carbohydrate origin), the result of overfeeding, especially when combined with constipation; and explains the good results of a stricter *régime* on these grounds.

### *Treatment.*

A consideration of the fact that headache is a symptom and not a disease renders it clear that proper treatment presupposes the recognition of the cause producing the headache. If the cause be amenable to treatment headaches will be banished, or, at least, diminished in frequency and severity.

In the graver conditions associated with severe intra-cranial pain, it will have been gathered from what has been said that active measures must be taken—trephining, either to remove a cerebral tumour or simply as a decompressive measure, and lumbar puncture in uræmia, etc. In cases of severe headache in the young subject, especially when worse at night, the possibility of cerebral syphilis should never be overlooked, and mercury and iodide should be given a thorough trial.

With increased blood-pressure, with or without marked arterial disease, especially when, as is so often the case, it is associated with the combination of over-feeding, over-drinking, over-strain, sedentary habits, and constipation, excellent results accrue from moderation in food and drink habits, from purgation, and from increased physical exercise. A course of iodides and the direct vaso-dilators, such as nitro-glycerine, is sometimes very efficacious. In migraine, for some unknown reason, paroxysmal vaso-motor phenomena occur from time to time, the constant repetition, indeed, being the factor the presence of which constitutes the disease in its accepted sense, in the same way as in the somewhat nearly allied disease, epilepsy, it is the repetition of fits which justifies the diagnosis of epilepsy. Limitation of diet with adequate exercise yields very good results.

The isolated headache is often attributable to dietetic errors, and preventive treatment consists in the avoidance of such. For the relief of the actual attack drugs are indispensable, and they are too well known to require mention. Many of the modern analgesic drugs are so efficacious that there is perhaps too great a tendency to their indiscriminate and repeated use, to the neglect of a thorough investigation of the general mode of life, and the elucidation of the real factor or factors constituting the deviation from the normal, on which alone scientific treatment depends.

*June 8th*, 1908.

* "Arterial Hypertonus and Arterio-sclerosis: Their Relations and Significance," 'Lancet,' June 1st, 1901.

THE Medical Golfing Society will hold its Tournament on June 18th at the Guildford Golf Club. Competitors must notify their entry by June 16th to Dr. Theo. Hyslop, Hon. Gen. Sec., Bethlem Royal Hospital, S.E.

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## PRELIMINARY NOTE ON A SPECIMEN SHOWING THE CHANGES ASSOCIATED WITH CHRONIC CONSTIPATION.

By ERNEST W. HEY GROVES, M.S., F.R.C.S.,
Assistant Surgeon, Bristol General Hospital.

THE patient was an old woman, æt. 78 years, who for many years had been an inmate of a workhouse. She had never suffered from any acute illness, and actually died quite suddenly in one of the ordinary wards, and not in the workhouse infirmary. She had been deformed with lateral curvature so long that the fact that the hip was dislocated and the seat of a large chronic abscess had escaped observation. Her body was brought to the Medical School in February, 1908, and it was not until the end of March that I discovered the condition of the abdominal viscera, and I then made inquiries from the Nurse Superintendent about the condition of her bowels. It is evident that this had been her chief conscious complaint, because even after this lapse of time the nurse could remember perfectly that she had had trouble about them.

But curiously enough the nurse's recollection was chiefly of diarrhœa, but she remembered that the patient had always been "bothering to have pills," but that the diarrhœa had been so troublesome they had had to restrict her diet, especially vegetables, and feed her chiefly on milk. This was for about six weeks before her death.

The external configuration of the body showed well-marked right scoliosis, dorsal dislocation of the right hip, with a huge abscess over the outer side of the buttock and thigh. On opening the abdomen the viscera exhibited some enteroptosis, that is to say, the hepatic flexure of the colon, the lower margin of the liver, the pylorus, and the lower borders of both kidneys were on a level with the posterior parts of the iliac crests; but since the ribs were touching the iliac crest this deformity was evidently more skeletal than visceral.

The most conspicuous object in the abdomen was the distended colon in its ascending and transverse portions, which varied in circumference from eight to ten inches, being two to three inches in diameter, the whole being densely filled with hard fæces. The cæcum was five inches long and two inches in diameter. Just above the right iliac crest the ascending colon and its hepatic flexure described a complicated loop which is six inches long if unravelled, containing five distinct kinks something like **W**, except that the various limbs do not all lie in the same plane. The kinking was maintained by the gut being bound together by serous membrane, which was supple and transparent like peritoneum, and it was by a direct continuation of this membranous adhesion that the fundus of the gall-bladder was tied down to the colon and duodenum. The transverse colon was fourteen inches long and about three inches wide, and presented a marked kink towards its central part, which occupied the cavity of the pelvis. The splenic flexure is in about the normal position and the descending, iliac and pelvic colons were quite natural in size and appearance and quite empty.

The stomach was very remarkably distorted in a manner which presents an exaggeration of the normal curves and divisions. A marked subdivision between cardiac and pyloric chambers existed, the former consisting of a globular cavity about five inches by four inches when distended, and the latter forming a canal which exactly resembled the small intestine, except for its thicker walls. At the junction of the two parts of the stomach was a well-marked notch on the great curve, and below this the pylorus formed a **U**-loop, each limb of which was three inches long, and was only half an inch from its fellow. This very remarkable kinking of the pylorus appeared to be caused by the direct drag of the loaded transverse colon pulling upon it by the gastro-colic omentum.

The third part of the duodenum lay across the second lumbar vertebra, and there is a sharp bend between the first part which runs nearly vertically up, and the second part which runs nearly vertically down.

The gall-bladder was rather contracted, and contained three stones in its cavity; thin membranous adhesions attached its fundus to the hepatic flexure of the colon and to the duodenum. The appendix was three and a half inches long, and was adherent in the cavity of the pelvis, the tip being actually attached to the right Fallopian tube.

The specimen thus illustrates the following points associated with the dilatation of the colon and chronic constipation:

(1) A matting together of the limbs of the colon by adhesions.

(2) A marked pathological kinking of the colon at the hepatic flexure and at the middle of the transverse colon, and an exaggeration of the natural kinking at the splenic flexure.

(3) A dragging down of the pylorus.

(4) A kinking of the antrum pylori.

(5) Biliary lithiasis with adhesions of the gall-bladder.

(6) Adhesions of the appendix and uterine appendages.

(7) A slight degree of enteroptosis.

*June 8th*, 1908.

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# THE CLINICAL JOURNAL,

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## CLINICAL LECTURE

ON

# A CASE OF DESCENDING TESTIS.


By C. MANSELL MOULLIN,

Senior Surgeon to the London Hospital.


GENTLEMEN,—One of my old teachers, for whom I always had, and shall have, the greatest respect, once said to me that if you wish to succeed either in teaching or in practising your profession you must be dogmatic, definite, and positive in all you say. It is certainly true that examiners do not like a candidate who has doubts; and private patients always believe in the man who believes in himself. They argue, and their position is quite intelligible, that a man who is not positive does not know; and, naturally, they prefer the man who gives them a definite opinion at once. But to-day I want you to put aside dogmas, and to consider some of the cases in the wards without prejudice, accepting nothing that is not capable of proof, and regarding everything with a sceptical frame of mind, no matter what name may be attached to it as authority, unless it is clear that the explanation fits in with all the facts of the case so far as they are known to us at the present time. Above all, I wish you to remember that calling things by another name does not constitute an explanation, even though you make use of words of prodigious length. Some such subterfuge from honest thought may be excusable at times when the public persist with unreasoning questions, but care must be taken that we do not unwittingly deceive ourselves.

The case I wish to discuss with you is that of a boy with an undescended testicle. He is just æt. 12 years, and well developed for his age. His right testis has never come down. One month ago something made its appearance in his groin, and now, when he strains or coughs, what is evidently

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the testicle comes done through the inguinal canal, and just emerges through the external abdominal ring, slipping back again at once. There is no hernia. Why has the testis not come down before? Why is it coming down now? and what is to be done for it?

Now, gentlemen, the number of reasons that have been assigned for non-descent of the testis is almost legion. Shortness of the vas, shortness of the vessels, intra-uterine peritonitis, contraction of the external ring, imperfect formation of the inguinal canal, the length of the mesorchium, an imperfect gubernaculum, spasm of the cremaster, and the size of the epididymis are a few of them. What proof is there that any one of these is the real reason? I have operated upon many of these cases, and I have scarcely ever been able to persuade myself of the value of any one of them. The vas, it is true, is often too short, but is this the cause or the consequence? Did the testis fail to come down because the vas was too short? or has the vas failed to grow because the testis did not come down? The latter is at least as reasonable as the former, and there does not appear to be any just cause for selecting the one view in preference to the other. So with the mesorchium. It is very often unduly short, but that again is no evidence that the shortness is the cause. The size of the external ring is equally valueless. If the testis has never come through it, and the cord has never occupied it, the ring may very well be smaller than it should be, but this is no proof that its diminished size prevented the descent. So it is with them all. The very simplicity of these so-called explanations is their attraction, but it does not follow that they are correct. One and all, if you notice, presuppose that the difficulty is a mechanical one. The testis has to travel a certain distance in the course of its development. It does not do so. Therefore, there is a mechanical impediment which prevents it. It may be so, but this is a decided assumption, and as I cannot help thinking, an assumption that is not warranted by the facts.

Now first, in considering this question, I wish to put upon one side all those cases which for want of a better name are sometimes spoken of as wandering or aberrant testes. For some reason or other they have left the well-established and recognised route. They may have gone down into the thigh, or into the perineum, or even into other localities; there is some additional factor here, some other cause. They are not simply cases of non-descent, the problem which I wish to consider to-day, and so for the present they must be put upon one side.

In man the normal time for the testis to come down into the scrotum is the eighth or ninth month of fœtal life—the left one first, the right shortly before birth. In some animals the rule is the same. In a few, including some of the most highly organised mammalia, the testes never come down at all. In the majority they do not come down until the period of sexual maturity is approaching. and in a good many they only come down during the period of sexual activity. For the greater part of the year they remain in the abdomen, and are comparatively insignificant in size. With each recurring sexual period they increase enormously in bulk and descend into the scrotum for the time. Then, at the end of the period, they go back into the abdomen and resume their former size and quiescent state. In such as these it is obvious that the descent of the testes, their increase in size, and their assumption of functional activity are simultaneous events regulated by the same cause.

Now in man there are the same three events as there are in these animals, only they are not, as a rule, simultaneous. The testes descend before birth, while they still retain their infantile proportions. The increase in size and the assumption of functional activity take place later. The origin of this difference is not difficult to understand. It is dependent upon the assumption of the erect attitude by man, and the consequent necessity for the early closure of the inguinal canal. Clearly it would not be for the advantage of the race if the tunica vaginalis remained widely open, leading vertically down from the bottom of the abdominal cavity until sexual maturity had been attained. A tendency to the same condition may be noticed among the old world apes. The events are the same, only the time relations are altered; and every now and then you meet with an exception, perhaps a reversion, as in the case of our patient to-day, in whom the three events are all taking place simultaneously.

Non-descent of the testes in man in the vast majority of instances has nothing to do with any of the mechanical causes which I have enume-

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rated, or with any others of a similar description. It is simply the persistence through childhood of a condition that is normal at a certain period of fœtal life, and is normal in certain classes of animals. Development has been arrested, just as occasionally the development of the upper lip or of the palate is arrested, so that hare-lip or cleft palate results. The arrest may be complete. In these individuals the testes retain throughout life their infantile character, size and position, and never become functionally active, so far at least as the production of spermatozoa is concerned. In a few, but very few (I have only known three instances of it) development progresses up to a certain point and then undergoes a process of retrogression. The testis in these cases develops normally and descends at the right time; and then ascends again into the abdomen and stays there. In others again the arrest, instead of being complete, is only partial. In these the testes descend a variable distance into the inguinal canal and sometimes become functionally active, sometimes do not. And finally, as in our patient of to-day, development may be arrested for a time, and then, at puberty, begin again. Just when it is beginning to assume its adult proportions, increasing in size at an enormous rate, the testis in this boy is beginning again to move down towards the scrotum. Within a month it has already come down the whole length of the inguinal canal, and there is no evidence as yet that it has stopped. The testis is behaving in this boy in the way that it does in many animals, and in the way presumably it would have continued to behave in the case of man, had not the assumption of the erect attitude rendered its earlier descent advisable for the good of the race. In what proportion of cases of retained testis this renewal of descent occurs I cannot say. I am afraid it is only a minority, and not a large one, although I have met with a good many. But I have no hesitation in saying that it occurs in a sufficiently large number to deserve to be taken into consideration, and it certainly has a very important bearing upon the treatment.

Given the case of a boy under fourteen years of age with a testis in the inguinal canal, the treatment depends upon whether there is a hernia present or not. If there is a hernia and the child is over one year old the hernia must be dealt with, and the testis with it. Such a hernia will not be cured by a truss. If it could not be prevented from coming down while the child was an infant in arms, thoroughly under control, it is not likely to be prevented by the same means when the child is able to run about. Some other plan must be adopted, and the only other plan is operation. But if there is no hernia and there never has been a hernia, the testis should be left and be given the chance of further development. I admit that there are certain risks. The testis, while in the inguinal canal, is always liable to injury. It may become twisted upon itself and strangulated. And there is always a potential hernial sac. The tunica vaginalis remains open in every case of undescended or partially descended testis. But so far as the testis itself is concerned the risk of injury or torsion is not a very serious one, not sufficient to justify immediate removal, and the liability to hernia, under these conditions, is not much greater. If no hernia came down during the first year of infancy it is not probable that any will come down during childhood. Two things are necessary for the descent of a hernia: one is a patent tunica vaginalis, and the other is a sufficient length of mesentery. Now, at birth the mesentery is so long that the small intestine can reach the bottom of the scrotum without any dragging. But after birth the length of the child's body increases so much faster than that of the mesentery that in a little while the bowel can scarcely touch the internal ring. The tendency, therefore, for the bowel to enter the inguinal canal steadily diminishes, and, as a matter of fact, strangulated hernia after infancy and before puberty is rare. I maintain, therefore, that if no hernia has ever been down, and the testis remains in the inguinal canal, it is worth while running the risk in the hope that at puberty when the testis begins to develop once more it may once more begin to descend.

At puberty, if the testis is still in the inguinal canal and shows no sign of moving onward, something must be done; what this should be depends upon the condition of the testis. If it is well developed and the cord is of sufficient length, or can be made of sufficient length to allow of the testis being transplanted into the scrotum, this may be done with a reasonable prospect of success. There is some hope that such a testis will maintain its nutrition, perhaps grow still more, and become functionally active. But if the testis still retains its infantile character transplanting it into the scrotum, supposing it can be done, is of no use. The testis has not failed to develop because it is retained in the inguinal canal. It is retained in the inguinal canal because it has failed to develop, and it is absurd to think that moving it on will make it develop. Such a testis must be removed and the inguinal canal closed.

*June 15th*, 1908.

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# DIAGNOSIS AND TREATMENT OF FRACTURES OF THE LONG BONES.*

Mr. A. Pearce-Gould. Mr. W. G. Spencer.
Mr. A. E. Barker. Mr. Robert Jones.
Mr. W. Arbuthnot Lane. Mr. Lynn Thomas.

(*Concluded from p.* 135.)

Mr. Lane next turned to the consideration of a few cases out of many showing the satisfactory results of operative treatment of fractures malunited after treatment by the orthodox measures.

Fig. 9.—Before operation.

The first in this class that he showed was a case of fracture of the humerus, which occurred at birth, and had been treated by manipulation and splints, with the result that the fracture was malunited to such an extent that the fragments were joined at a right angle, and there was paralysis of the musculo-spiral nerve. This unsuccessful issue of treatment by splints and manipulation was clearly indicated in Fig. 9, and the fortunate termination in consequence of the operative interference with the malunited fracture was exemplified by Fig. 10. It was interesting to note that the operative treatment further enabled the surgeon to free the musculo-spiral nerve from the callus in which it was embedded, and to cure the paralytic condition.

Fig. 11 showed an ununited fracture of the shaft of the humerus. The bone had been fractured more than a year before. It was treated by splints and massage with the result that the fragments did not unite. She was then operated on

Fig. 10.—After operation.

on the Continent without success; the arm remained useless because of the non-union of fragments. Musculo-spiral paralysis followed this unsuccessful operation. The ununited fracture was exposed, and the fragments so arranged as to obtain perfect apposition, which was retained by plates and screws. The paralysis disappeared, and the patient obtained a useful arm. The result of the successful operation was shown in Fig. 12.

Fig. 13 showed a precisely similar sequence except that there was no paralysis. Primary treatment by splints, etc., resulted in non-union, while

* A discussion at the Medical Society of London.

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the operative measures which had been adopted at a later date were equally unsuccessful. In each case the failure to secure union was consequent on the inefficient means employed to obtain it. Accurate coaptation of fragments and their fixation by means of screws and plates was well illustrated by Fig. 14.

Fig. 15 showed the result of treatment of comminuted fractures of the radius and ulna by means of manipulation and splints. The injury resulted from the crushing of a laundry machine. The arm

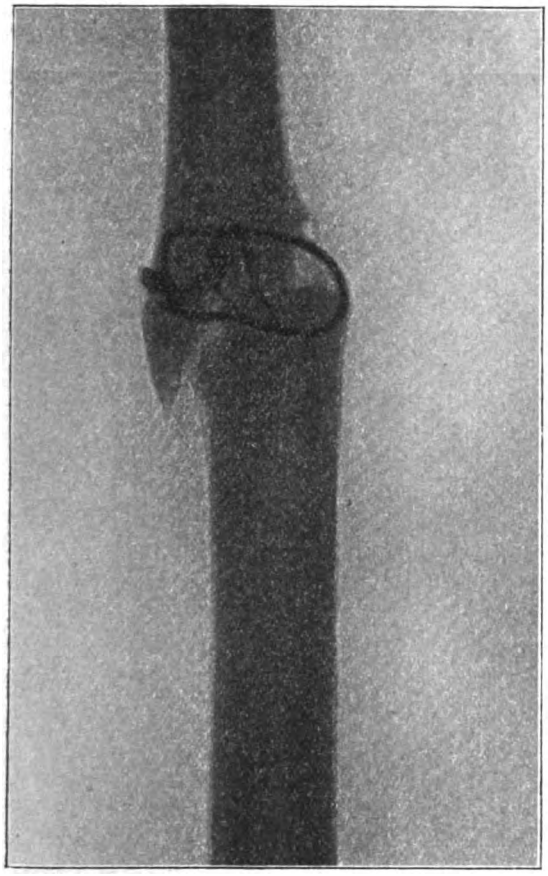

FIG. 11.—Ununited fracture of humerus.

was perfectly useless, pronation and supination being absent.

The bones were exposed and divided in various planes, the fragments being subsequently fastened together by means of screws and plates in the best possible position in the circumstances.

Fig. 16 showed the condition some months after this operation, when the functional capacity of the arm was gradually improving.

Fig. 17 showed the deformity following on a fracture of the radius which had occurred in the same situation on three occasions within a year. The functional activity of the arm was considerably reduced. The fracture had received the closest attention on every occasion. Fig. 18 illustrated the result of operative treatment. The deformity was rendered trifling while the functional activity was very materially improved.

The next case was that of a boy, æt. 11¾ years, who had a fracture of the radius and ulna. This was treated by splints and massage, with the result that he was not able to pronate or supinate

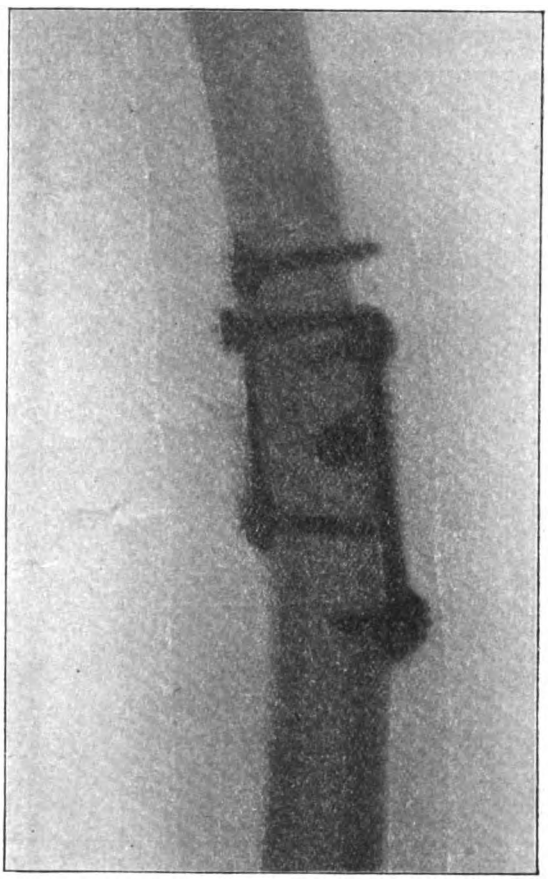

FIG. 12.—Result of operation by Mr. Lane.

the forearm. Fig. 19 showed the deformity of the radius and ulna responsible for the disability present. The bones were divided in four separate planes, and the fragments were brought into the best position possible, and were so retained by plates and screws. The boy was very pleased with the functional result, the anatomical aspect of which was displayed by Fig. 20.

Fig. 21 showed ununited fractures of the radius and ulna in a boy, æt. about 18 years. He had been under the care of a surgeon of great repute, and had been treated in the usual way. The arm

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was perfectly useless. Fig. 22 illustrated the result of operation, perfect functional activity being restored, the anatomical form being also normal.

Figs. 23 and 24 represented a precisely similar condition to the preceding case. In this case also the patient was in competent hands, and had received most careful treatment by means of manipulation and splints. He now had a perfectly useful arm.

Fig. 25 illustrated the result of treatment of a fracture of the femur by manipulation, splints and massage. The patient was in good circumstances, and was able to avail himself of excellent surgical advice and treatment. He had three inches shortening, which, together with the altered rotation of the lower fragment, rendered locomotion exceedingly difficult and ugly.

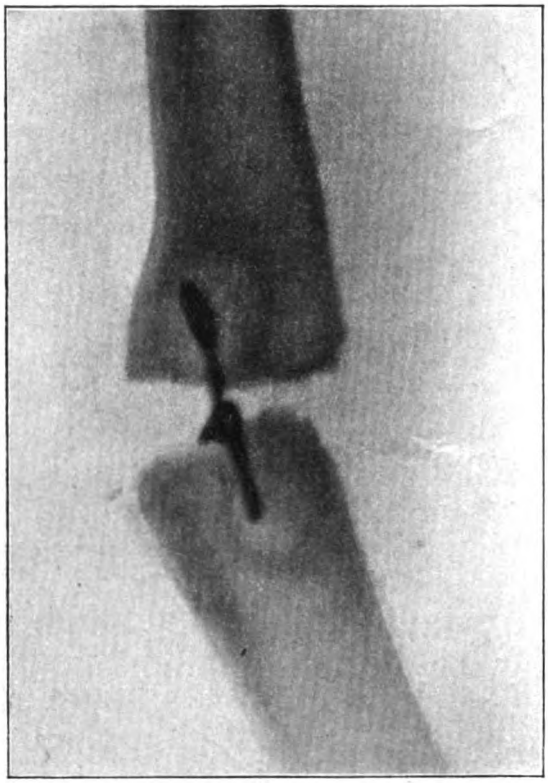

FIG. 13.—Ununited fracture.

The fracture was exposed and the bone was divided in two planes, with the result shown in Fig. 26. The axes of the fragments were almost perfectly continuous and the shortening was comparatively trifling. The patient could now get about in comfort.

Fig. 27 illustrated the result of treatment by manipulation and splints of a case of fracture of the femur. The leg was three and a half inches shorter than its fellow, and union had taken place at such an angle that the patient was quite unable to walk.

Fig. 28 showed the condition after operation, which took place seven months after the receipt of the injury. There was then a shortening of one inch, which was readily compensated for by an elevator half an inch in depth, while the femur was functionally perfect.

The next photograph shown represented a femur which had been operated on because of mal-union. Mr. Lane said he had exposed the ununited fracture, cut suitable surfaces, and secured the fragments in accurate apposition. The femur was now functionally perfect. The result was shown in the radiograms handed round.

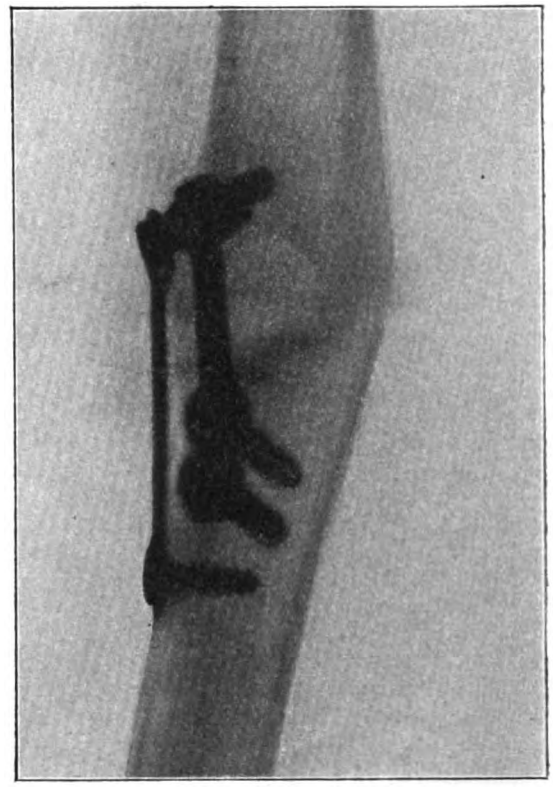

FIG. 14.—Result of operation by Mr. Lane.

The next two photographs showed the result of treatment of a fractured femur by manipulation and splints, with complete disability, and the result of subsequent treatment by operation.

Fig. 29 showed the unfortunate result of the treatment by manipulation and splints of a fracture of the femur in a boy, æt. 9½ years. He was operated on in the Hospital for Sick Children four and a half months after the receipt of the injury. The resulting condition was shown in Fig. 30.

Fig. 31 illustrated a case which Mr. Lane regarded as the barometer of the surgery of the present day.

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The patient came up to consult him because of disability and deformity resulting from a fracture of the femur, which had been treated unsuccessfully by manipulation, splints, etc. He urged operation. The man said before consenting he must have another confirmatory surgical opinion. He was unable to get another opinion in London in favour

Fig. 15.—Before operation.

Fig 16.—After operation.

of operation. Everybody told him that an operation was unjustifiable. It was asserted by some that it was impossible to overcome the overlapping of the fragments since the nerves and arteries could not be stretched. He still remained in his crippled condition.

The next photograph showed an ununited fracture of the tibia and fibula of a Welsh miner, æt. 33 years, who had been unsuccessfully treated by splints and manipulation. Mr. Lane was obliged to remove a considerable amount of bone in order to obtain perfect axial continuity of fragments, so that the limb was left two inches shorter than its fellow. When the shortening was compensated for the patient was able to resume his occupation. The result of this operation was shown in the skiagram handed round.

Fig. 32 represented an ununited fracture of the tibia and fibula in a man, æt. 23 years. He had been treated unsuccessfully by manipulation and splints for seven months.

The successful result of operation was shown in Fig. 33. Axial continuity of the fragments of the

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tibia was obtained at the necessary cost of some shortening, which was readily compensated for.

Fig. 34 showed the disastrous result of treatment of a fracture of the tibia and fibula by manipulation and splints for eleven weeks. A plaster case was then applied and worn for ten weeks. He was then told to walk, but was re-admitted and again

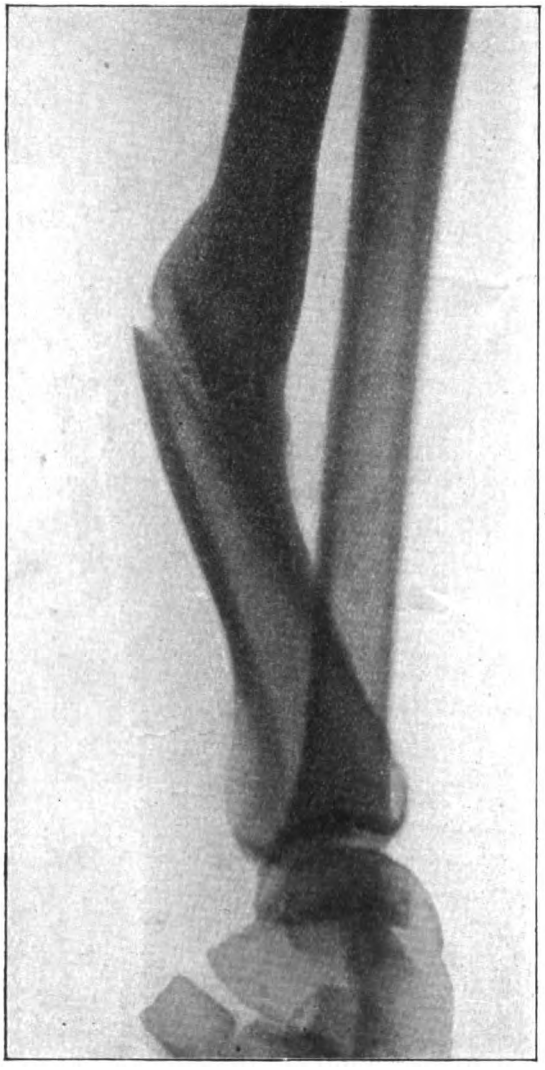

Fig. 17.—Before operation.

treated with splints for eleven weeks and with a plaster case for seven weeks. He left the hospital on crutches, and had only been able to follow his occupation since very imperfectly and for very short periods of time. He suffered very severe pain in the ankle-joint, and especially where the fibula impacted on the front of the facet on the astragalus. This was much aggravated by a strain which brought him under Mr. Lane's observation.

Fig. 35 showed the result of operation by means of which the patient was enabled to lead an active life with a reasonably useful leg.

Figs. 36 and 37 represented antero-posteriorly and laterally the method that Mr. Lane considered best in the majority of cases of fracture of the patella. He employed a plate with two or more

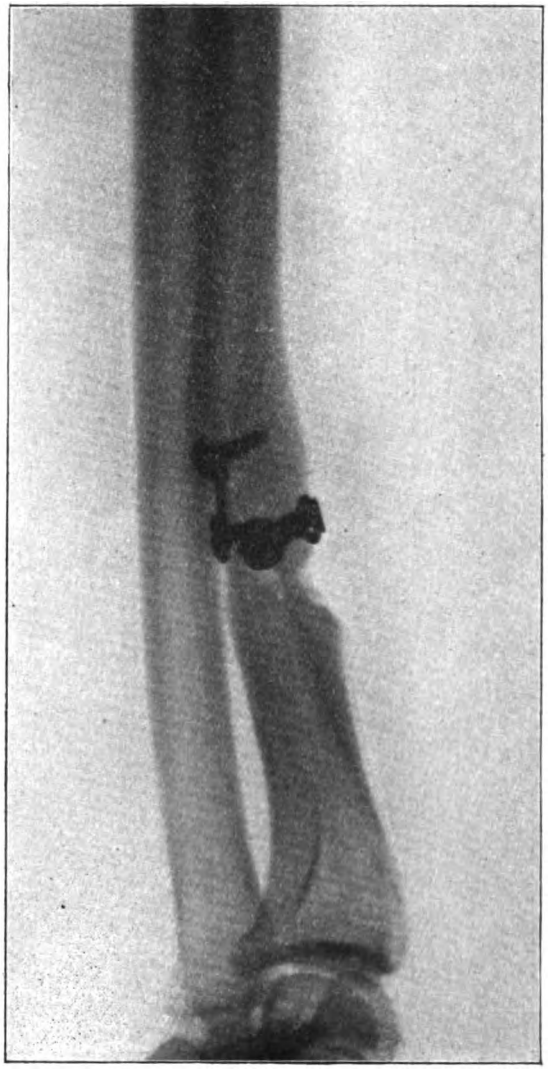

Fig. 18.—After operation.

screws. The screws used for this purpose were half an inch long and were threaded up to the head. Such screws were more tenacious than ordinary screws, and were used when it was not possible to perforate and grip a distal compact tissue. These screws and plates had many advantages over wire, since they could be introduced with the greatest accuracy and facility into the fragments, whose broken surfaces were retained in the most accurate

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and firm apposition by means of suitable forceps. The hold upon the fragments by the screws and plate was more tenacious than that of wire. No projecting knot or eminence was left beneath the skin, since the heads of the screws were flush with the thin plate, which itself was buried beneath the periosteum, etc., of the patella.

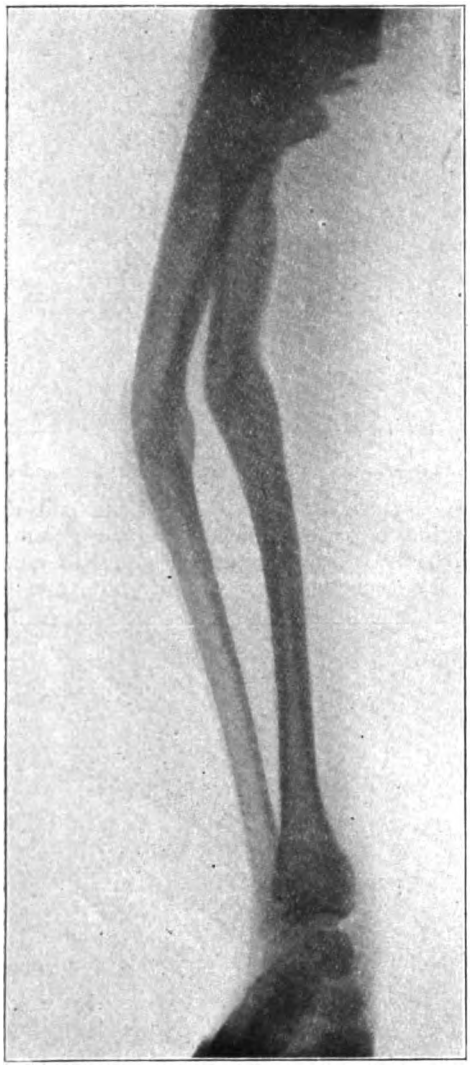

FIG. 19.—Before operation.

In conclusion he said that he was exceedingly sorry to differ absolutely from the eminent surgeons who had already spoken, but he appealed to the judgment of the profession on the subject.

Mr. W. G. SPENCER said he thought it was impossible for bony union to occur in the cancellous short bones and ends of long bones unless the fractured surfaces were in exact position, and in that connection one should recall the controversy about the neck of the femur, Sir Astley Cooper denying that such union could ever take place by bone. That was disproved by some specimens in the College of Surgeons. Observation by means of X rays of such fractures which had been either wired or had pegs put in them showed that there was not atrophy of the bone in connection with the peg or

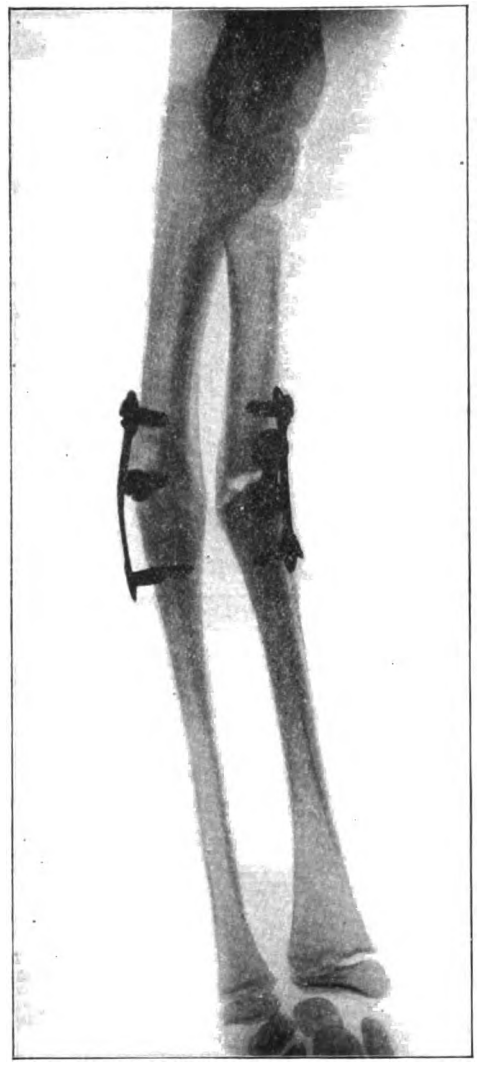

FIG. 20.—After operation.

wire; therefore he thought that wiring through the cancellous tissue rested on a different basis to pegging or wiring through the middle of long bones, where the callus was largely formed from the periosteum. In the skiagrams of these shown by Mr. Lane one could see that atrophy took place along the grasp of the wire, or of the screw, and that was not a new observation. Mr. Gould's opening of the discussion on the fractures of long bones

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included the ends, and he mentioned Colles's fracture also. Therefore he (Mr. Spencer) thought

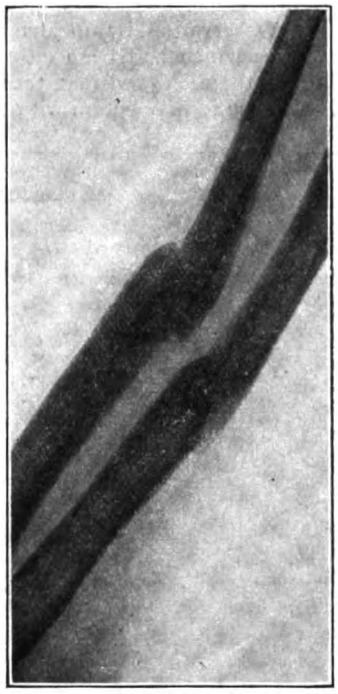

FIG. 21.—Before operation.

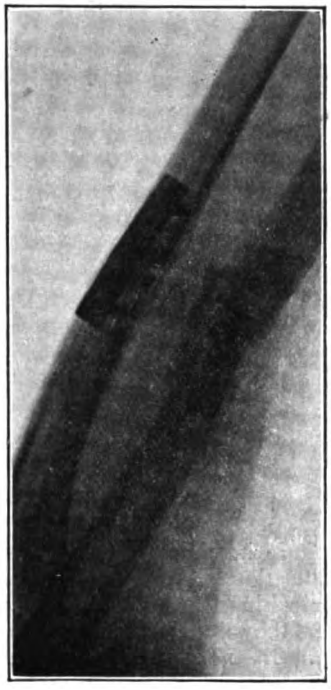

FIG. 22.—After operation.

it was necessary to go back to the difference in the formation of callus. In the union of the shafts of long bones, where that was largely periosteal, there was not the necessity for the exact apposition which Mr. Lane had advocated, because there was a chance of bridges from the periosteum forming the external callus, which was absent in the bones

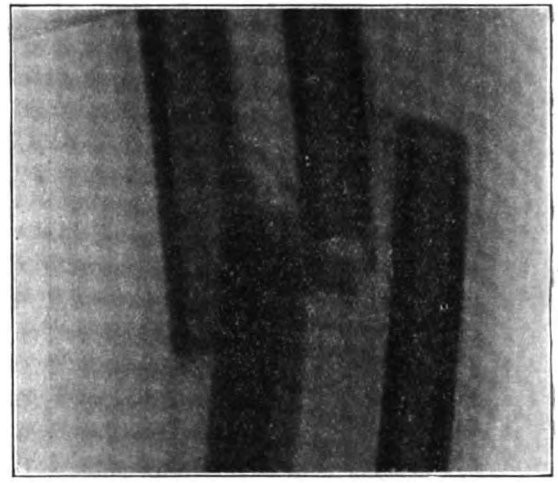

FIG. 23.—An ununited fracture of the radius and ulna.

of the patella or skull. Mr. Lane had shown skiagrams of cases which, for some reason, had been badly treated by others and had come to him for secondary operations, but one must

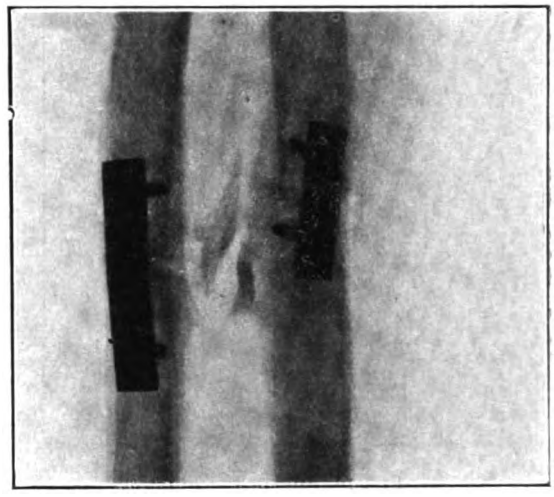

FIG. 24.—The results of operation on fracture shown in Fig. 23, the replaced fragments having been secured by screws and plates.

distinguish those from the cases spoken of by Mr. Gould, although Mr. Lane's results were very brilliant. If the union of the shafts of long bones could be brought about without the insertion of a foreign body between the bones, that was surely better than putting in such objects. Mr. Bryant

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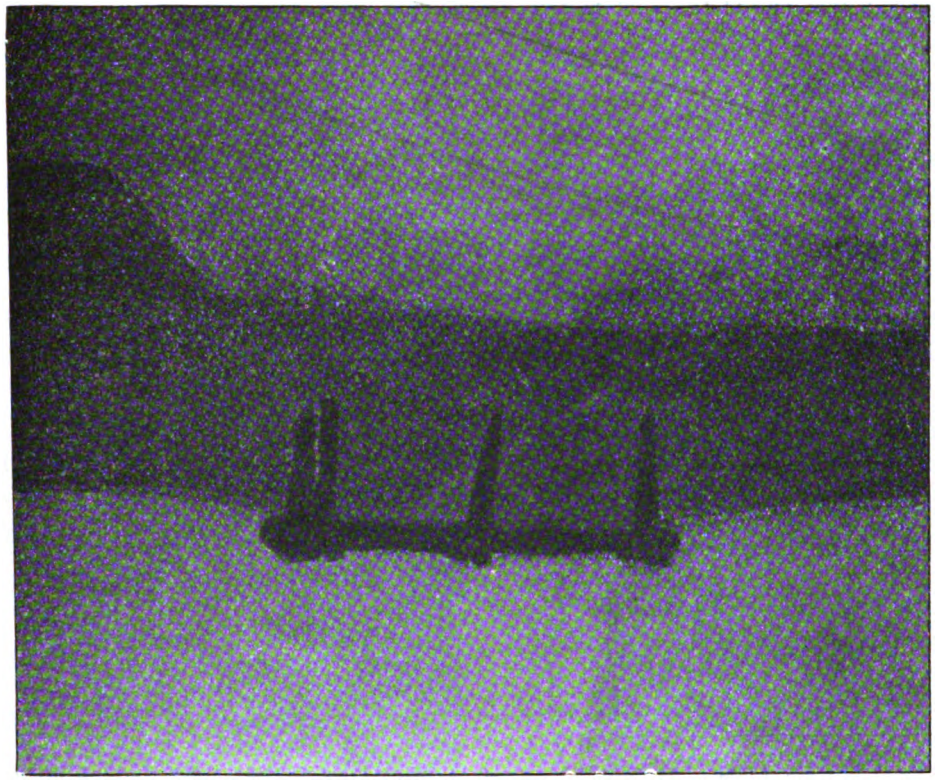

FIG. 26.—After operation.

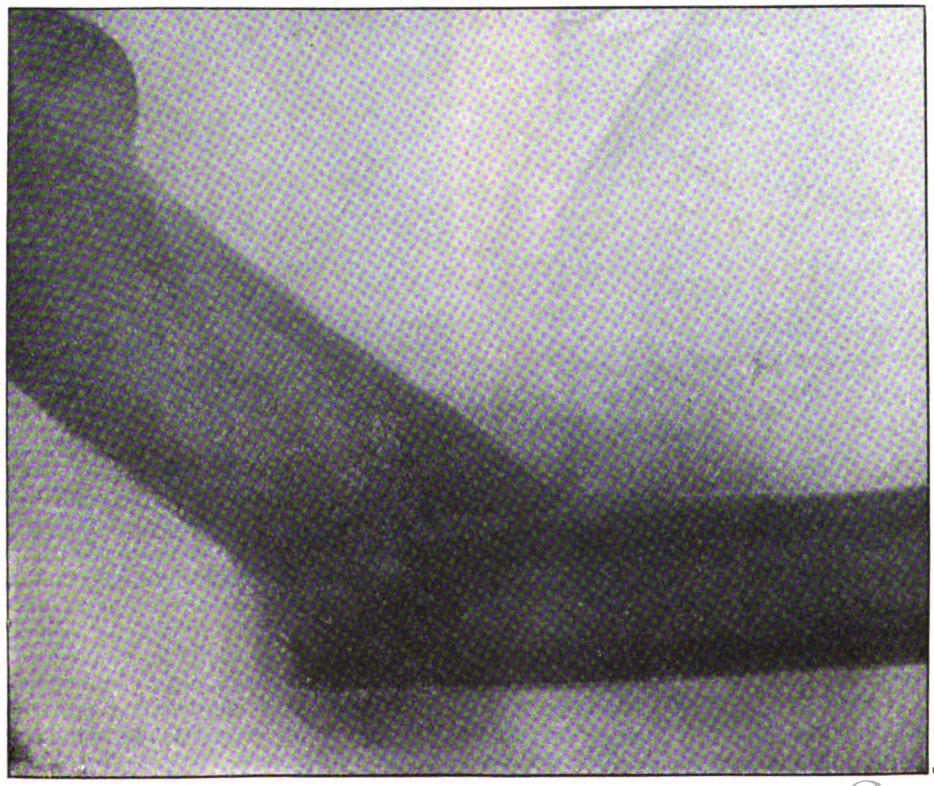

FIG. 25.—Before operation.

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showed long ago that one of the great means of overcoming displacements in the common fracture

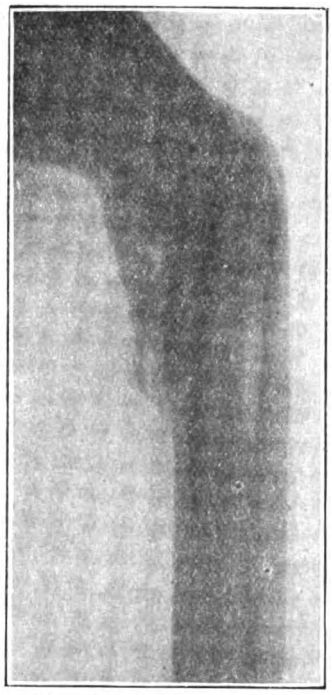

FIG. 27.—Before operation.

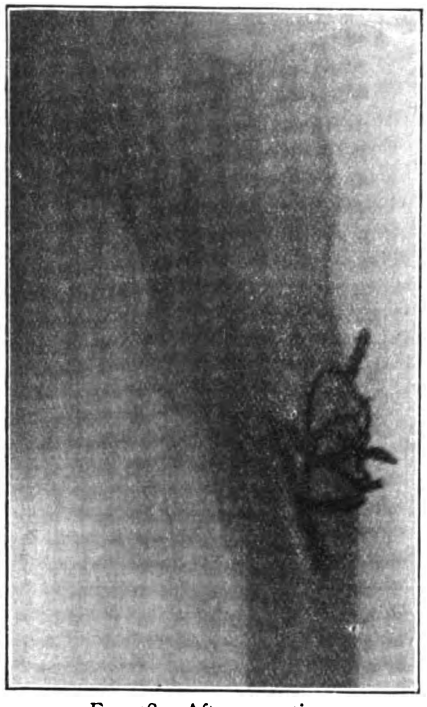

FIG. 28.—After operation.

of the leg was the direction of the tendo Achillis, and why should these be wired or pegged if they would engage after manipulation? The occurrence of atrophy along the course of the wire or peg seemed against the use of a foreign body.

Mr. ROBERT JONES (Liverpool), complimented Mr. Pearce Gould on his interesting and practical paper, with most of which he agreed. He thought surgeons should be very guarded in their language when they laid down rules on the management of fractures now that the general public were so ready to take actions at law. The

FIG. 29—Before operation.

public should be made to clearly understand that perfect end-to-end apposition is only rarely secured, and that fractures were not "set" in the popular sense of the term. They should be impressed that different surgeons had their own special ways, which, while differing from one another, led to recovery. Some surgeons, like Lucas-Championniere, thought that a little movement of the fractured ends of bone aided union, and he practised massage and ambulation. In his hands this special method had proved most successful. Other surgeons at home and abroad wire every fracture in order to obtain accurate

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apposition. In their skilled hands such cases do well. Some surgeons depended on splints almost entirely, and having mechanical aptitude did admirably for their patients. The knowledge that all these schools had able votaries should diffuse a spirit of toleration amongst us. It should prevent surgeons characterising the work of their colleagues as "wrong treatment"—such lack of loyalty did infinite harm to our profession. The public should be told the fallacies that arise from the distorted images displayed by Röntgen rays. Although Dr. David Morgan and Mr. Jones

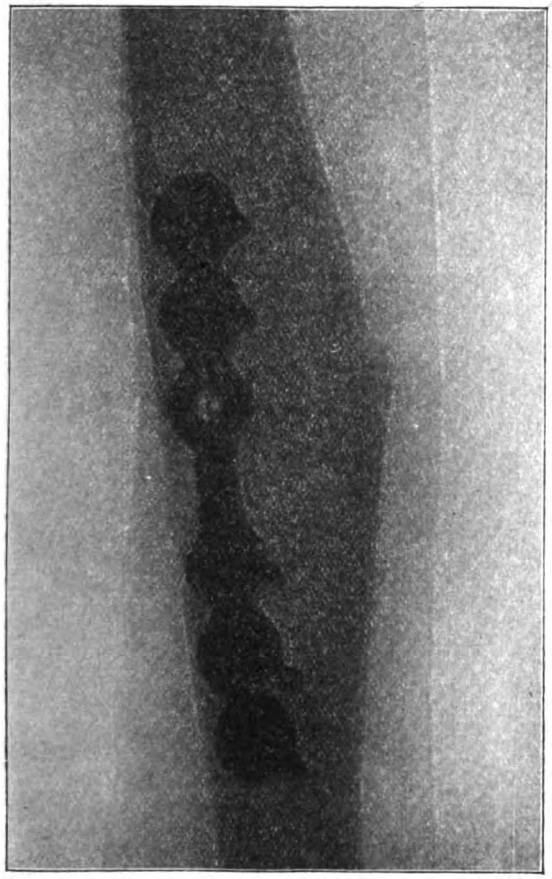

FIG. 30.—After operation.

took many radiographs a day they constantly differed as to their interpretation. Although the X rays had been of service as an aid to classification they had practically brought about no revolution in the treatment of fractures. Personally he made his diagnosis, and in order to educate his diagnostic power he employed radiography. It was deplorable to think of the education of the student of to-day, who rarely troubled to make a diagnosis, but laid implicit trust in an X-ray plate. The public should be told two facts: First, that with a most accurate apposition may be allied a useless limb; and secondly, with a very ugly bony union good function may exist. This should not prevent the surgeon from getting the best apposition he can. The bony injury often proved the least serious to the limb, and was only one element of often serious injuries to the limb. Injuries to vessels, muscles, and nerves were never considered by the public, who speak of a broken leg as a thing of small account. He constantly had patients come to him brandishing angrily a radiograph and blaming their practitioner if the apposition were not accurate. In his opinion the two sources of failure in the treatment of fractures were due to tight circular constriction by bandages, and erroneous deflection

FIG. 31.—Mr. Lane's barometer of present-day surgery.

of body-weight on to the fractured bones. He frequently saw the ill-effects of keeping fractures of the upper humerus and injuries to the elbow fixed in splints, and since he had discarded their use his results were better. In fractures of the upper end of the humerus he merely used a sling, and kept the arm under the shirt. In injuries and dislocations of the elbow he supinated and completely flexed the joint. Ischæmic palsy, the treatment of which Mr. Jones described, was due to pressure from within or from without, and produced the same pathological changes as were to be found following frost-bite or a forgotten tourniquet. A correct alignment was far more important than accurate apposition. This was especially the case in fractures of the lower limbs. A proper alignment prevented a harmful deflection of body-weight. The defect in function was generally due to angular deflection at the point of fracture or to injuries of the soft parts. An example of this was seen in the disabilities which

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follow a malunited Pott's fracture. Where correction of the eversion restored the function it was never necessary to bring about an end-to-end apposition; all needed was an osteotomy of the fibula; a wedge from the tibia, and to restore the alignment. All surgeons of experience knew the cases which give them trouble; they were usually those—whether apposition be good or bad—where the bones had united at an angle. In old fractures of the thigh overlapping was of little account compared with varoid or valgoid deflection,

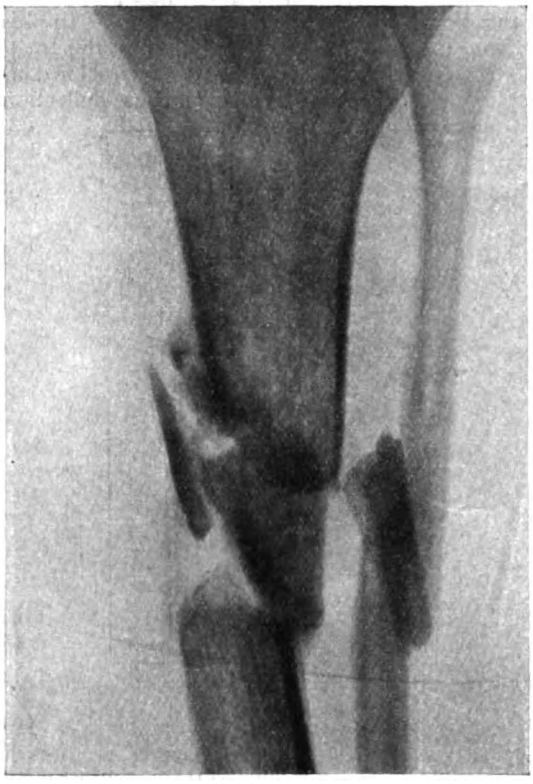

FIG. 32.—Before operation.

and instead of cutting down and wiring such fractures it was sufficient to do an osteotomy and correct the deflection. Mr. Jones emphasised the necessity for studying the mechanics of the after-treatment of fractures, of which, generally speaking, the hospital surgeon, as such, had no experience. Massage and movements were necessary adjuncts of treatment, but active movements were of more value than passive, and many an injured elbow had been worked stiff by the vigorous Swede. In tender joints it was most important that the movements should be gentle and gradual, to avoid the evils of friction.

Mr. C. B. KEETLEY referred to a paper which he read fifteen years ago on the prevention of shortening in the shafts of the long bones after fractures. The method was to make a pin-hole in the skin opposite each fragment and then put a pin straight through the flesh and the bone; an important detail was to keep those pins a long way from the point of fracture. He showed two cases of fractured thigh which had resulted in a practically perfect result, *i. e.* there was no shortening, one in a man, the other in a boy, and he had been practising that method ever since. He was now quite sure that it was an absolutely simple and safe method—

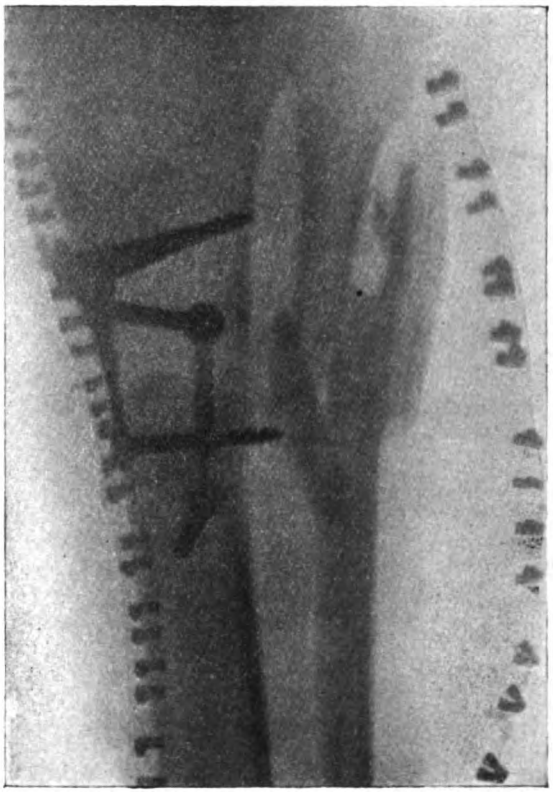

FIG. 33.—After operation.

that is to say, safe in the hands of any surgeon in the room. For years it had been his practice to make use of mechanical extension, but he had not made use of it in the two cases which he showed. He made mechanical extension by means of a Bessemer steel frame and screw force. It might sound complicated but was not so in reality, and no person who had got into the habit of using such a frame would resort to manual force, with the aching arms and backs of assistants. Mechanical extension bore the same relation to manual that typewriting did to ordinary writing. None of his surgical friends and colleagues had shown any feverish desire to be beforehand in imitating his device, but he did not expect that in fifteen years,

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so that he was not disappointed. But he felt sure it would be plagiarised, more especially the principle of keeping the pins apart. But it had been copied in a country where there was more intellectual activity than in England, namely, by Mr. Clayton Parkhill, of Denver, who used his (Mr. Keetley's) pins divided into thirty-two separate pieces, some as screws, some as screw-drivers. It had also been re-invented in Belgium, and he believed Mr. Lynn Thomas had some experience of the method. But

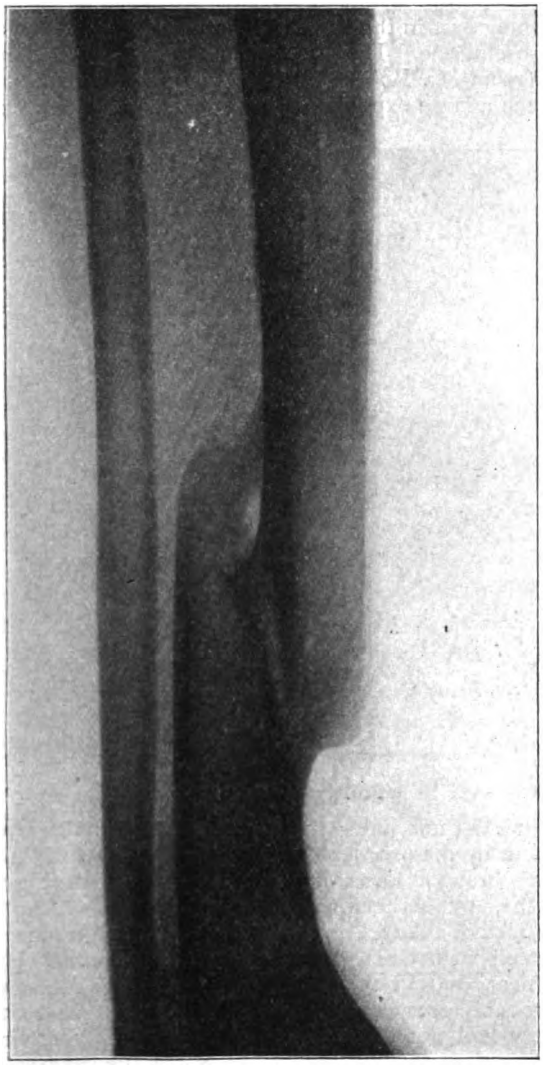

FIG. 34.—Before operation.

in Belgium it was not applied subcutaneously, but a large incision was made.

Mr. LYNN THOMAS (Cardiff), said he had come a long way in order to hear the discussion on a subject in which he took more interest than perhaps he might have done but for circumstances outside his control. Many surgeons belonged to no school at all, and he was glad to hear Mr. Robert Jones emphasise the fact that they should belong to no particular school, because cases varied so much that they had to be treated according to the light, experience and judgment of the surgeon. Fifteen years ago he saw Champonniere treat his cases simply by massage, and he thought it an extraordinary method, because he had never heard of it. Everyone saw disasters in surgery, but always, as Mr. Gould had said, in

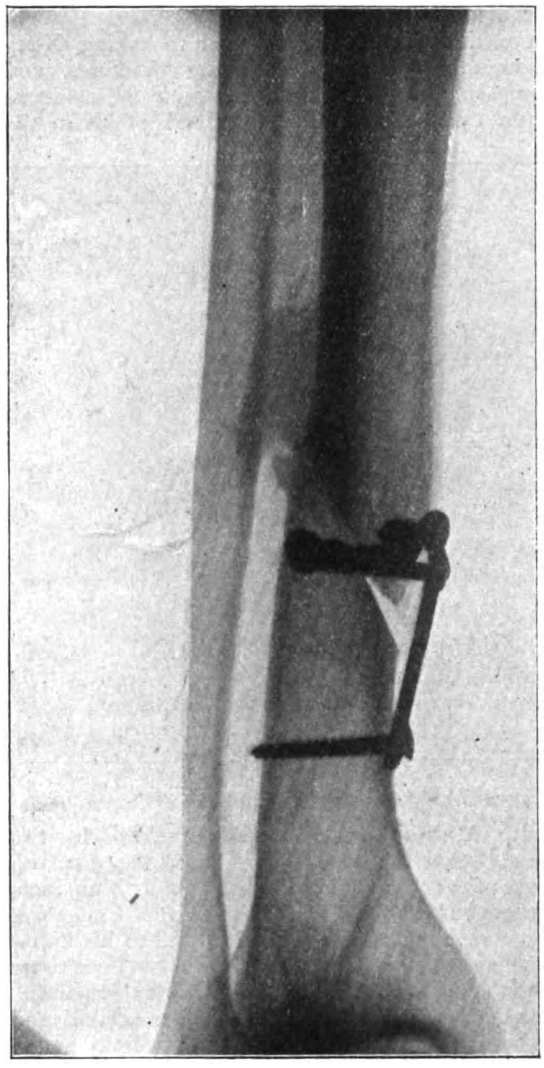

FIG. 35.—After operation.

other people's practice. There were at present in the country a good many cases of simple fractures which, if they had been left alone, would probably not be walking perfectly now, but at all events they would have had their limbs with them. No case should be operated upon unless it was quite certain that non-operative procedure would not

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yield a satisfactory result. The use of X rays had made no difference at all to the line of treatment which he adopted in his own practice. He saw a large number of fractures, and had the privilege of being consulting surgeon to a hospital in which the surgeon-resident treated all his cases of fracture of the lower extremity for six weeks in splints, and fractures of the upper extremities four weeks in splints. And in some cases, especially in Colles's fracture, the results were very disastrous compared with the results of the treatment which Champonniere had recommended. He thought more time should be devoted to taking X-ray photographs of results of ordinary fractures, and comparing the functional result with the evidence given by the skiagram. A friend of his in the South African war fell and fractured his leg two inches below the knee-joint. There was displacement of the tibia and overlapping of half an inch. He was treated by a Fellow of the College of Surgeons, and came to London and to his knowledge went the round of half a dozen surgeons to try to get something done, as it was extremely painful. But he went to the non-operative school and nothing was done. He wanted his limb amputated. The deformity was shown by the skiagram to be as bad as in any of the pictures shown by Mr. Lane, and the result was extraordinary. So having an X-ray photograph of fractures a long time afterwards and comparing them with the functional result had given him a great deal more confidence in Nature than he had when he started his surgical career in Cardiff.

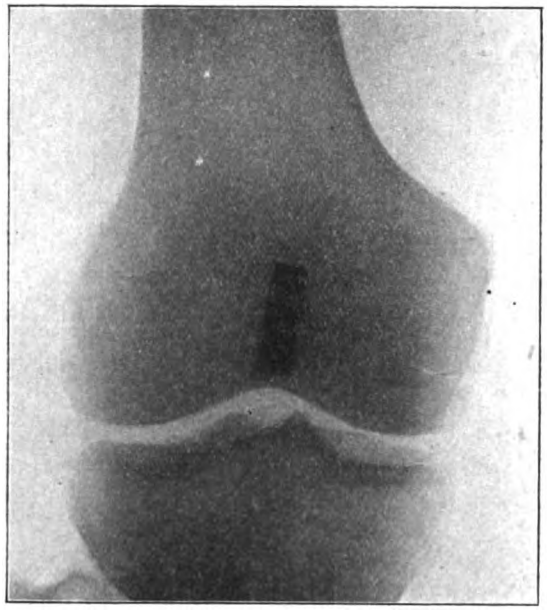

Fig. 36.—Antero-posterior view.

Dr. Silonville (Paris), said he had seldom been fortunate enough to hear such an interesting discussion. He had always advised his countrymen to come to London to hear about such things, and he felt more inclined to do so than ever now. In cases of fracture of the femur it had been the practice in France during the last fifteen years to have much more recourse to extension than was usual in England. He hoped to hear that extension was used in this country to a greater extent than it appeared to be by the discussion.

Mr. Gould, in reply, thanked members for taking part in the discussion and making it such a a useful one. He had been particularly interested in what Mr. Robert Jones had said, as he had had such a large experience. He presumed that Mr.

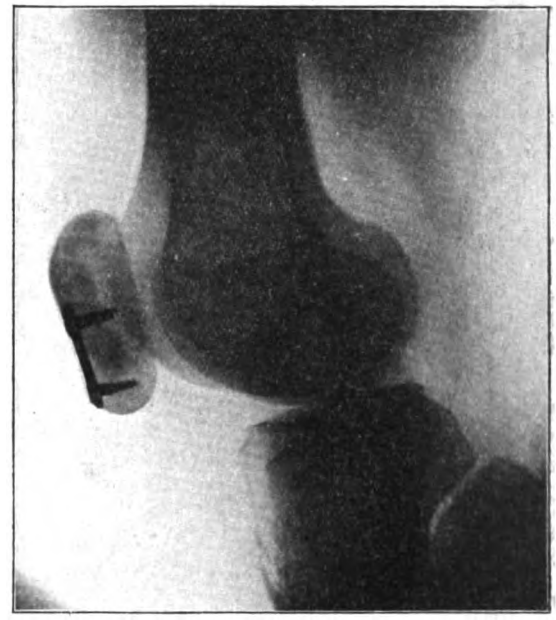

Fig. 37.—Lateral view.

Lane did not judge the results of the usual treatment by the excellent series of skiagrams which he had brought forward. Naturally, Mr. Arbuthnot Lane, by his eminence in the profession, had attracted a large proportion of cases of fracture which had been unsatisfactorily dealt with by others, and in the majority of the cases shown probably every one in the room would agree that they had not been treated successfully by other methods, and certainly operations in those cases were urgently called for. Mr. Lynn Thomas's case of serious deformity from fracture of the tibia, in which the patient was eventually able to take more exercises than most surgeons had the opportunity of enjoying, was very instructive, and Mr. Thomas's advice to take skiagrams of old fractures and compare that with the functional result was advice which surgeons would do well to follow.

*June 15th*, 1908.

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# SOME INTER-RELATIONS OF ABDOMINAL AND THORACIC DISEASES.*

By SIR WILLIAM HENRY ALLCHIN, M.D., F.R.C.P.,

Consulting Physician to the Westminster Hospital and to the Victoria Hospital for Children.

THE enormous developments and extent of our knowledge, the time required in the details of examination, more particularly for the practice requisite to become skilled in the use of instruments of precision, individual inclination, and lastly, accident, have combined to make us all specialists, some more, some less. The time has gone by for inveighing against the inevitable, and it only remains for us to be on our guard against some of the ill-effects that specialism engenders, whilst enjoying to the full the advantages which attend a concentrated attention on one particular line.

At the outset I would affirm my belief that by the special study of certain groups of diseases, whether the grouping be based on regions of the body, on particular organs or systems of organs, or on individual pathological processes, the accumulation of detail which such procedure has entailed at the same time furnishes the ground for taking a wider conception of disease in its manifold bearings, and thus tends to counteract the very evil that specialism may give rise to—a too restricted view of the bearings of disease as it affects particular organs or functions. In this way are we enabled, whilst thoroughly ascertaining the condition of heart, lungs or liver, brain or kidneys, which the perfection of physical examination and increasing precision of chemical investigation enable us to do,

* Introductory Address at the opening of the Post-Graduate Course, Mount Vernon Hospital for Diseases of the Chest.

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at the same time to look not only at these organs themselves, but at the general effects, mechanical, vital or toxic, which their morbid conditions determine, and thus that specialism so called should by the greater knowledge it provides at the same time ensure a truer and more scientific aspect of disease.

I am to-day addressing those who are mainly concerned with the investigation and treatment of a wide range of disease limited only in its restriction to one region of the body, and whilst others who have preceded me in the honourable position I for the moment occupy have out of the fulness of their knowledge directed your attention to some special affection of the chest or its contents, I would rather prefer to devote this occasion, which your courtesy has afforded me and for which I desire to express my very high appreciation, to the consideration of some of those relationships, both structural and functional, which exist between maladies as they affect the two principal regions of the trunk, chest and abdomen—regions which in their earliest condition form but one common cavity, but that by their subsequent anatomical separation with specialisation of function come to be characterised by certain pathological features of great clinical interest, more particularly in that zone where the boundaries of the two divisions are in contact. Moreover, the fundamental differences in the nature of the functions performed by the thoracic viscera from those carried out by the organs in the abdomen are associated with such differences in the methods requisite for the investigation of these processes, whether in health or disease, as to suggest a distinction in the leading characters of the morbid states as they affect these two divisions of the trunk, and to go far to justify a specialisation in their study. For whilst the problems presented by the heart and lungs, in their abnormal as in their normal working, are predominantly physical, it is in connection with them that the methods of physical examination, more especially percussion and auscultation, have been brought to their highest pitch of perfection, whilst the organs subserving the functions of digestion and excretion are much less capable of physical investigation since their workings are so largely chemical in character. It is, of course, true that certain alterations in size and resistance of the contents as well as the presence of abnormal growths or collections of fluid in the belly are ascertainable by the trained application of the senses, which constitutes physical examination; it is the sense of touch—the tactus expertus, and still more the tactus eruditus—which is far more important and helpful in the investigation of the abdomen, and percussion, though of some, is of distinctly less value, as auscultation is scarcely applicable at all. It is, I say, these distinctions in the modes of inquiry and of obtaining the evidence upon which the diagnosis is in great part based that determine in a measure a different cast of mind with different lines of thought, according as the problems that are presented to the view of the physician deal mainly with thoracic disorders, or those in connection with the abdomen, and afford, I venture to hope, a subject which has some interest to those who will find a common ground in the neighbourhood of the diaphragm.

No doubt there are many morbid states of each division of the trunk—chest and abdomen—in which the other region is not involved either by the occurrence of any structural disease, or, indeed, of any symptomatic manifestation except so far as the general health is affected by the existing malady. Many illustrations of this statement will readily occur to you. As examples I might refer to an ordinary attack of acute bronchitis in which the symptoms are almost or entirely referable to the chest, and any discomfort that may be located in the abdomen will be only such as would follow from the strain of the muscular parietes from excessive coughing. Nor is it usual to meet with any abdominal symptoms in connection with acute endocarditis or with pericarditis unless the effusion be considerable, nor, indeed, with most cases of aneurysm above the diaphragm. Similarly, many conditions of gastro-intestinal disease, such as ulceration and even new growths, of hepatic, pancreatic and renal disorder, may run their entire course without any appreciable affection of heart or lungs until within the range of impending death.

In contrast, however, certain maladies of one region inevitably lead to disease in the other, or are directly responsible for symptoms in the other without the production of actual disease of structure, so that the chest may be involved secondarily to the abdomen, or conversely abdominal disease may be directly consequent upon intra-thoracic disorder, it being also

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remembered that coincident involvement of both regions is possible.

Before proceeding to consider these groups of morbid conditions in their pathological and clinical bearing, it may be well briefly to have before us the structural mechanism, as it may be termed, upon which this mutual involvement depends. Concerning the *parietal components*, integuments, muscles and bones, it will suffice to refer to the modifications that may arise in the respiratory acts by impaired mobility of the abdominal muscles or the exaggerated movements of these same structures that may follow an undue fixity of the lower thoracic region. An enormous increase in the subcutaneous fat may also impede the breathing, and is a factor of no small importance when difficulties of respiration already exist from pulmonary or cardiac disease.

Of far more interest are the *vascular connections* between the two regions of the trunk, and of these the venous communications are much the more extensive and important. The essential object of these anastomoses is to ensure an efficient return of blood to the heart should there be any obstruction to either of the caval trunks or their primary branches. Under normal conditions the communicating vessels are small, and in parts of their course the blood-flow takes place indifferently upwards or downwards so as to reach the right auricle by either vena cava, but should there be an interference with the circulation through either of these main vessels, then these communicating channels develop enormously, sufficient if need be to maintain the entire blood-stream.

The disposition of these vessels is both parietal and visceral, between which also free but small and numerous communications exist. The former are situated both superficially, *i. e.* in the integuments, and deeply within the trunk. In this way a direct connection is maintained between the axillary and innominate veins, and so with the superior cava on the one hand and the iliac veins and inferior cava on the other, by means of the intervening internal and external mammary, superficial and deep, superior and inferior epigastrics, which in turn are joined in their course by branches from the intercostal, lumbar and circumflex iliac veins, and that open themselves into the saphenous, and so to the internal iliac. Owing also to the free communication between the vena azygos and the parietal intercostal and lumbar veins, and these last again with the iliacs and inferior cava, the two cavæ are by another route brought into connection, and it is a little significant that this is largely affected by the adult representative (vena azygos) of a vessel that in the earliest stage of embryonic development brings back the greater part of the blood from the posterior regions of the body (vena cardinalis).

It is well to picture in our mind's eye this ordinarily unimportant system of vessels, but one that under some circumstances may be called on to play a most important part in the blood return. Thus may the whole venous drainage of the lower limbs and abdomen ultimately reach the heart by way of the superior cava, as much of that from the upper half of the trunk and head may find its way by the inferior cava.

Nor is this all. Owing to the venous return from the chylopoietic viscera being interrupted in its course by the interposition of the portal circulation, a further opportunity of obstruction occurs, the consequences of which are to a certain extent compensated for by certain communications between this portal distribution and the general systemic venous system, this anastomotic arrangement at the same time contributing in a small degree to the communication between the upper and lower cava, the main features of which have just been alluded to. By means of branches between the superior hæmorrhoidal vein and the hæmorrhoidal plexus, and between this and the internal iliac, the portal and systemic systems are united, and the same takes place at the juncture of the coronary veins of the stomach with the œsophageal vessels, which *via* the azygos vein establishes continuity with the superior cava, both these being arrangements whereby portal obstruction may be relieved, though not unattended with risk from over-distension and rupture of their communicating plexuses, with severe and even fatal hæmorrhage. Less frequently remembered, but still from their exceeding number both effective and important, are the minute vessels in the areolar tissue and peritoneal folds about the liver which connect with the portal branches in that organ, and also with the phrenic and azygos, and the still more numerous connecting venules that establish communication between the vessels of the kidneys, pancreas and other organs with the

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lumbar radicals in the post-peritoneal connective tissue. Better known from their superficial position, and the frequency with which they are seen to be greatly distended are those branches (par-umbilical) which extend between the surface of the liver and the epigastric vessels in the abdominal wall, the course of which has been previously traced.

Concerning the arterial supply to the chest and abdomen and their contents little need be said as bearing on our present subject. Certain anastomoses take place between branches of the thoracic and of the abdominal aorta of which that between the terminals of the internal mammary branch of the subclavian with the epigastric from the internal iliac on each side is one of the most direct. For the most part the viscera and the parietal structures of the trunk are separately supplied by special branches from the aorta, but in the post-peritoneal connective tissue, and to a lesser degree in the sub-pleural tissue, numerous minute communications take place between terminal twigs of the bronchial, hepatic, renal, supra-renal, pancreatic, duodenal, cæcal, and certain of the colic arteries, with others from the intercostal, phrenic, lumbar, circumflex iliac and other of the parietal vessels. In this way a very extensive interconnection is maintained between the visceral vessels and those which supply the wall of the trunk, and the relief to congested organs by bleeding or counter-irritants over the adjacent integuments is probably thus in great part explained.

The *lymphatic connection* between the thoracic and abdominal cavities is closely associated with the anatomical question as to how far the peritoneal and serous sacs are to be regarded as being essentially enormous lymph spaces with free communication with the lymphatic vessels. It has long been taught, as the result of the observations of von Recklinghausen, Arnold, Klein, and others, that between adjacent endothelial cells of the peritoneum and pleura there exist definite apertures known as "stomata," by which direct communication is established between the peritoneal cavity and the subperitoneal lymphatic vessels through the intermediary of the subserous lymph spaces. The more recent researches, however, of Muscatello and Kolossow, which have been confirmed by MacCullum, disprove the existence of these openings as other than artifacts, and go to show that the endothelial layer, except at the openings of the Fallopian tubes, is absolutely complete. Slender minute prolongations or closed diverticula of the peritoneum have been described as dipping vertically or obliquely among the fibrous bundles of the central tendon of the diaphragm, entirely lined by endothelium, and here it is that by means of phagocytes an extensive transference of solid particles such as micro-organisms takes place from the peritoneal cavity to the lymphatic channels—a special characteristic that is shared in by the great omentum. But whether there be actual continuity through open perforations or not there is certainly effective drainings from the peritoneal sac into the diaphragmatic lymphatics, some of which are received by the post-sternal glands which lie along the internal mammary artery, inasmuch as these glands are quickly implicated in acute peritonitis, the infecting organism having been found in them even before the evidences of inflammation of the membrane are apparent. In this connection the rapid and extensive absorbing power of the serous membranes should be remembered, the withdrawal of fluid being effected by both blood-vessels and lymphatics, which their close anatomical relation to the membranes makes possible. It is supposed that the latter vessels are chiefly concerned when the effusion is moderate in amount and gradually poured out, the former playing the greater part when the accumulation is excessive, and there is reason to believe that so far as the peritoneum is concerned the greater part of the absorption is brought about at the diaphragmatic surface.

The superficial lymphatics of the anterior, lateral, and posterior areas of the thoracic and abdominal parietes above the level of the umbilicus, together with the vessels from the upper limbs, drain almost entirely into the anterior axillary (or pectoral) and posterior axillary (or sub-scapular) glands, which in turn communicate with the thoracic duct and right lymphatic duct. Those from the lower part of the abdominal wall, including the superficial branches of the peri-umbilical network, pass to the inguinal, and thence *via* the iliac glands to the thoracic duct. Free anastomosis takes place between the vessels of the two sides of the body.

The deep lymphatics of the thoracic walls and parietal pleura are received by the intercostal and

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by the internal mammary or sternal glands, from all of which efferent branches extend more or less directly to the thoracic duct. The corresponding vessels in the abdomen also reach the thoracic duct through the intermediary of several series of deeply situated glands between the peritoneum and transversalis fascia, viz. external iliac, those accompanying the deep circumflex iliac and deep epigastric arteries, internal and common iliacs, lumbar and aortic. The deeper vessels from the upper part of the abdominal wall and corresponding peritoneum pass to the internal mammary glands; as also do those from the anterior part of the diaphragm and anterior diaphragmatic glands; whilst from the same part of the diaphragm and from the rest of that structure lymphatics drain into the several groups of mediastinal glands and so to the thoracic and right lymphatic ducts. It is to be noted that the lymphatics of the diaphragm are disposed in thick networks beneath the pleura and the peritoneum respectively, but whilst free communications take place between these two layers of vessels which perforate the muscle, there is but slight communication between the right and left plexuses.* The lymph vessels of the heart and lungs with their serous coverings, of the thymus, œsophagus, and other structures pass to the mediastinal glands, the middle set of which include those termed "bronchial."

Associated with most of the abdominal viscera are groups of lymphatic glands similarly named, which receive the lymphatics from the substance of the organs. From thence efferent vessels pass to the glands about the aorta, and so to the receptaculum chyli and thoracic duct. From the kidneys, supra-renals, testes, and ovaries the lymphatics pass direct to the aortic glands. Owing to the peculiar investment of the liver by peritoneum, and the formation by that membrane of certain so-called ligaments, certain of the superficial lymphatics of that viscus drain into the diaphragmatic glands, and so reach the internal mammary glands, and also anastomose, with the special network that surrounds the umbilicus.

"It is well known that the supra-clavicular glands sometimes become enlarged subsequent to the appearance of malignant conditions in the abdomen and the mediastinum, and more particularly subsequent to affections of the œsophageal portion of the stomach. The course along which the infective material travels in these cases is not yet known, for as yet no efferent vessels have been traced to these glands from the mediastinum."*

In the distribution of the lymphatic vessels to the viscera they are usually so arranged as to form a capsular or peripheral plexus, whilst other branches penetrate the substance of the organ. Between the former and the parietal lymphatics anastomoses occur, comparable to what is met with in the blood vascular system.

The integuments and parietes of the trunk are *innervated* by the thoracic or dorsal nerves, excepting quite the uppermost and lowest regions, which derive their supply from the cervical and lumbar plexuses respectively. The upper six dorsal nerves are distributed to the thorax, whilst the lower six are thoracico-abdominal in their distribution and all overlap. The diaphragm, which is mainly supplied by the phrenic nerves from the cervical plexus, receives, however, some branches from the lower intercostals, which "contribute to the sensory innervation of the margin of the muscle and possibly also slightly to the motor innervation."† Among the terminal filaments of distribution of these dorsal nerves are those which reach the subpleural and subperitoneal connective tissue, and it is upon these branches that the sensibility of the serous membranes, pleural, pericardial, and peritoneal, depends. By means of the rami communicantes—white and grey—the spinal nerves are brought into connection with the ganglia of the sympathetic trunk. Through the medium of these rami, white or medullated fibres from the spinal ganglion cells enter and pass through the sympathetic nerves to their ultimate distribution; these constitute the splanchnic efferent fibres, whilst others derived from the ventral (anterior) root of the spinal nerves end in the sympathetic ganglia, and are known as the splanchnic efferent fibres. The grey rami, which are mainly composed of sympathetic fibres and are non-medullated, pass to their distribution along with the anterior and posterior primary divisions of the spinal nerves, some, however, terminating in the spinal ganglia. It is by means of the splanchnic branches that the cerebro-spinal system is brought into communication with

* 'Morris's Human Anatomy,' 1907, p. 724.

* 'Text-book of Anatomy,' edited by Professor Cunningham, 1906, p. 913.

† 'Morris's Human Anatomy,' p. 424, 1907.

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the viscera, to which these branches are ultimately distributed, either directly or through the intermediary of the various collateral and terminal ganglia and plexuses. Branches of the vagi also enter into the several thoracic and abdominal plexuses, and are supplied thence and even directly to the viscera, more particularly heart, lungs, and stomach.

By the cerebro-spinal (somatic) nerves to the chest and abdomen and their contents the voluntary muscles are governed and sensory impressions are conveyed, whilst the regulation of the unstriped and the cardiac muscles and the glandular tissue is affected by the autonomic (sympathetic) system, but though distributed with them the fibres capable of giving rise to sensation properly belong to the somatic system (Langley).* It is due to the intimate connection taking place through the rami communicantes between these two systems of nerves that various reflex motor disturbances, such as cramps and rigidity, may be induced, and that cutaneous pain and tenderness of visceral origin may be experienced in definite and constant areas corresponding to the spinal segment or segments connected with the affected viscus. It is this which constitutes what is known as reflected pain, whilst by referred pain is meant that which is due to irritation of a nerve or one of its branches, and is felt either over the entire peripheral distribution of the same nerve, or at the termination of some other branch to that irritated.

There remains to be referred to the *diaphragm*, that musculo-membranous septum between the two divisions of the trunk that separate them one from the other and yet serves as a close connection between them. I am apt to think that the extent to which the diaphragm rises into the chest is not always sufficiently remembered, and that in the course of our physical examinations we do not always realise that the muscular dome, and with it, of course, the subjacent structures, notably liver and stomach, normally rise up to the level of the fourth interspace in the mammary line just below the nipple on the right side and rather below that on the left, and that in some conditions of abdominal disease the encroachment on the thoracic cavity may even extend up to the second right interspace. The several serous membranes are very closely attached to its two surfaces, with the interposition of a very slight amount of subserous connective tissue, which, in the subphrenic situation, is free from fat. Among the various structures which pass through the diaphragm it is important in the present connection to remember the post-peritoneal tissue, which becomes continuous through the aortic opening with that occupying the posterior mediastinum, and thus becomes part of an extensive layer that, lining the entire trunk, also extends into the neck, and limbs. In the abdomen it also passes between the layers of the mesenteries and omenta, being distributed with the blood-vessels, with the outer coats of which it is connected.

Clinically, the most important functional relation between chest and belly centres round the movements of the diaphragm in connection with respiration. What an effective share the contractions of this muscle take in enlarging the thoracic cavity, and in consequence of the diminished pressure therein determining the entrance of air to the lungs, and greater inflow of blood into the large veins, is only fully appreciated when morbid conditions arise on either side of the diaphragm that interfere with its free action. To the portal circulation this action of the respiratory pump is all important, since by it this circulation is largely maintained, and it is in connection therewith that the earliest indications of cardiac difficulty may be apparent. How often, too, is it that some mechanical interference with the proper movements of the diaphragm, together with the diminished available respiratory area due to the upward invasion from the abdomen, are responsible for a fatal ending to many a chronic condition of heart or lungs where death has been warded off by a gradual adjustment of the morbid conditions, but where it was impossible to cope with this last straw, as it were, afforded by an impeded diaphragm. When the abdominal fulness is due to fluid the difficulty may, for a time at least, be overcome; but when some enormous liver, or more serious still and quite as intractable, a flatulent distension with a little ascites is existent, it is most disheartening and almost humiliating to watch our patient succumb to the additional mechanical trouble that resists all efforts at relief.

(*To be concluded.*)

*June 22nd*, 1908.

* 'Brain,' vol. xxxvi, 1903.

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# HÆMATEMESIS: ITS VARIETIES AND TREATMENT.

A Clinical Lecture delivered at the London School of Clinical Medicine, Greenwich.

By SIR DYCE DUCKWORTH, M.D., LL.D., F.R.C.P.,

Consulting Physician to St. Bartholomew's and the Italian Hospitals; Senior Physician to the Seamen's Hospital, Greenwich.

GENTLEMEN,—Hæmatemesis is a symptom, and not a disease. When summoned to a patient of whom you know nothing, and who has brought up a large quantity of blood, it is not easy to be sure at first whether the bleeding has its origin above or below the diaphragm. The patient is often quite ignorant whether he has vomited or coughed up the blood. In cases of epistaxis and hæmoptysis blood may be first swallowed and subsequently vomited. Hæmatemesis may be small or large in amount. Bleeding from the gums, buccal, and nasal surfaces may be spat out, and suggest gastric hæmorrhage. Spongy gums in a foul and neglected mouth not seldom yield a watery, fœtid, and bloody fluid without any streaks. Patients sometimes clear their throats after taking cocoa, essence of beef, or after chewing tobacco and taking snuff, and these cause a stained expectoration simulating altered blood from some source. I have known clinical clerks to be deceived in this way. We are, however, more particularly concerned to-day with the graver forms of hæmatemesis, which indicate organic disease and a breach of the gastric or other alimentary mucous surface.

The two most frequent sources of this symptom are gastric ulceration, or erosion, and cirrhosis of the liver.

I discuss first, gastric ulceration and erosion.

The occurrence of vomiting of blood is always alarming to the patient. The amount of blood may be large, both fluid and in clots, and the vomiting may be repeated, so that the patient may within an hour or two be reduced to a state of profound anæmia. This condition may readily induce grave anxiety to the practitioner, unless he has reason to believe that the hæmorrhage is an accident in the course of a gastric ulcer. If the patient is a young woman the case is almost certain to be of this nature. It is now ascertained that bleeding of this kind is hardly ever fatal, but this fact is not so widely recognised as it ought to be. The hæmorrhage, with appropriate care of the patient, tends to cease in most cases. I lay stress on this point, for the occurrence of serious hæmatemesis is now rather too commonly regarded as a positive indication for immediate surgical interference, and an endeavour to ligature or excise the bleeding surface. I therefore, as a rule, deprecate any such measures, and am content to treat the patient on ordinary medical principles, and I expect to promote recovery under appropriate treatment. As I have said, these cases are most often met with in young women from fifteen to thirty years of age. We must now note that the bleeding may depend upon two varieties of gastric lesion: (*a*) An erosion, or patch of sore and excoriated mucous surface, yielding an active and progressive oozing of blood, which collects in the stomach and is vomited at intervals; and (*b*) a defined, acute, depressed ulcer, or ulcers, for these may be multiple, commonly situated on the posterior wall of the lesser curvature of the stomach, which has opened into an arterial branch in its progress. It is important to be aware of these distinct lesions, for, as we shall find, the former is of much more sinister significance than the latter or commoner variety.

The acute defined ulcer and the erosive oozing patch are what we have to deal with in young women. In the former we may meet occasionally with severe hæmatemesis which may recur several times. This bleeding tends to stop. In the erosive form we find severe hæmatemesis, which recurs, and is sometimes incoercible, leading to fatal anæmia. Ordinary measures may prove of no avail in these cases. A chronic ulcer of the stomach may be very large, and is generally single. We meet with these chiefly in men and in middle life. They may lead to serious bleeding, owing to involvement of large gastric vessels, and even sometimes, as I have seen, from opening up the splenic artery to which they have become fused.

In any of these forms of hæmatemesis it is usual to find that blood has passed on through the stomach, and escapes from the bowels, black and tarry-like, in the form of melæna. If the hæmorrhage is small in amount, it undergoes

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changes in the stomach, and is thrown up in the form recognised as "coffee-ground" vomit.

When repeated hæmatemesis, uncontrolled by ordinary measures, occurs in a young woman, we should suspect that we have to deal with the oozing erosive ulceration, and surgical interference must be duly considered before the patient becomes too blanched and collapsed. I have met in recent times with two cases of this nature. In one the erosion was discovered on opening the stomach, and the bleeding chaps or fissures were severally secured by fine ligatures. The hæmorrhage ceased at once, and sound recovery was the result. In the other, the source of the bleeding could not be found, and a fatal result from continuous oozing ensued. The erosion was probably high up in the lesser curvature, and could not be reached after as complete an eversion of the stomach as could be made.

Duodenal ulcer sometimes gives rise to hæmatemesis, but more commonly the hæmorrhage is passed along the intestine, and is detected by the occurrence of melæna.

We are not concerned now with the ordinary treatment of gastric ulcers, but it is important to urge an early recognition of this condition, and to secure absolute rest and appropriate diet for the patient, and where there is only a suspicion of ulcer, founded on the presence of some of the characteristic symptoms, it is always wise to treat the case for some time as possibly due to this lesion, rather than to some form of dyspepsia. Many cases of gastric ulcer never present the symptom of hæmatemesis, and the exact diagnosis is not always easy to establish.

I may mention one source of copious hæmatemesis which may depend on an unrecognised small aortic aneurysm, which bursts into the œsophagus and rapidly proves fatal.

Cancerous ulceration of the stomach is not often the source of free bleeding unless a large artery is opened up, such as the splenic. Small "coffee-ground" hæmorrhages may, however, occur in these cases.

I pass on now to discuss the second common cause of copious hæmatemesis, namely that arising in cases of advanced cirrhosis of the liver. This is a very different matter from that we have just considered. The hæmorrhage may be the first marked symptom to attract the serious attention of the patient. He may have had many plain indications of the disorder which has been in progress, and may not have sought advice for them. The general aspect of the body, and the history of dyspepsia, diarrhœa, and morning retching would at once arrest the attention and suspicion of any practitioner, and lead to examination of the liver. This organ would usually be found unduly firm and much enlarged. There would generally be no ascites. The mawkish alcoholic breath, icteric conjunctivae, and the presence of venous stigmata on the face, neck, and arms would also suggest the cause of these symptoms, and declare the patient to be suffering from chronic alcoholism. The history, not seldom, as related by the patient or his relatives, may be quite misleading, and especially is this the case if the patient is a female. No heed must be paid to asseverations of strict temperance in these cases. Many cases of hepatic cirrhosis arise in persons who have not really grossly abused alcoholic drinks. As I related to you in a recent lecture on this subject, the vulnerability of the liver to alcohol varies remarkably in different people.

The amount of blood vomited in these cases is sometimes enormous and startling. The patient may be bleached rapidly by it, and assume a cadaverous aspect. He is certainly in great peril, for the vomiting is apt to recur copiously, and sometimes proves quickly fatal, even within a few hours.

What has happened is a rupture of certain varices or dilatations of the gastric branches of the portal vein, and not infrequently this occurs in some engorged loops of the veins of the lower end of the œsophagus, which have gradually formed piles at this point. The whole portal venous system is congested, and relief comes by way of rupture at some point. The blood vomited is very dark, and much of it passes down the bowels as melæna.

Then patients often have small hæmorrhages in the earlier stages of cirrhosis, such as epistaxis, hæmoptysis, and bleedings from the small stigmata and the gums, this condition being one of cachexia, for cirrhosis is no mere local hepatic malady. The older physicians described this hæmorrhagic condition as due to "a dissolved state of the blood."

*The treatmemt of hæmatemesis.*—In cases of gastric ulcer absolute rest is to be enjoined. The alarm and unrest of the patient are to be

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allayed. Rest in bed on a hard mattress (with the head low and the feet kept warm), with perfect quietude is to be secured. Such bleedings are hardly important save as confirmatory evidence of any previously entertained suspicions as to the nature of the case. Large bleedings, as I have already stated, tend to cease in most instances, and rarely prove fatal in the case of acute depressed ulcers. The patient may promptly show signs of grave anæmia. Sometimes the effused blood is not ejected by the mouth, and passes by the bowel. Remember that bleeding is rarely the first recognisable symptom of a gastric ulcer.

The great point in practice here is to put nothing into the stomach for at least four or six hours. Any craving for cold drinks must be firmly resisted as tending to encourage more bleeding and vomiting. The first and best liquid to administer is whey, or more suitably, alum-whey, in half-ounce doses (prepared by adding a drachm of powdered alum to a pint of milk, nearly boiling, and straining through muslin). Later, albumen, beaten up with water and a little glucose (the *eau albumineuse* of the French codex) may be given in smal amounts, not too frequently. Afterwards, milk with one third of lime-water, or with the addition of liq. calcis saccharatus, Ph.B., ten minims to the ounce. Lactate of calcium five grains to the ounce may also be used. Cream and water is of service also a day or two later. The rectum may be washed out the day after the hæmorrhage with a small enema, and subsequently enemata of ordinary saline solution with a little glucose may be given as a nutrient. Large injections are undesirable as tending to provoke vomiting. Sanatogen is useful for this purpose, also the yolk of egg. It is advised to apply ice to the epigastrium, but I am not satisfied of its efficacy. Repeated bleeding has been checked by a twenty-minim dose of turpentine, rubbed up with albumen. Ergotin in one- or two-grain doses is employed sometimes, but I have no experience of its value. Adrenin, ten minims of one in a thousand solution, is given as a hæmostatic, and I have employed it, but I am not sure that it is quite free from objection, and I am not satisfied of its importance. As a matter of experience, with absolute rest for the patient and absolute non-interference with the stomach, I have found that these cases generally do well, and make good progress to recovery. There is no risk in a partial starvation for a few days.

It has been alleged that hæmatemesis may occur without any breach of the gastric mucosa. I am very doubtful as to this, and strongly suspect that in such cases there has been an erosive lesion such as I have described. These erosive patches are hardly detectable save for their bleeding during life on opening the stomach, and at a necropsy there may be absolutely no signs of them owing to digestive and post-mortem changes.

In hæmorrhage dependent on cirrhosis of the liver much the same treatment is to be employed as just described—absolute rest and quietude, and nothing is to be given by the mouth. The prognosis is always grave, and repeated vomiting of blood with a fatal issue is the common experience in these cases.

In cases of gastric ulcer which prove rebellious to prolonged medical treatment, or manifest signs of adhesion of the stomach to adjacent parts, the operation of gastro-jejunostomy is now proved to afford great relief and comfort to the patient. Careful feeding and a well-ordered life are necessary in all cases of gastric ulcer for many months after the lesion is believed to have healed. Unfortunately, it is often impossible to secure such management in the majority of these patients, who are seldom found amongst the well-placed classes of society. Hence we meet with relapses.

*June 22nd*, 1908.

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**Gastric Diagnosis.**—Fischer points out that the usual classic signs and symptoms and the most approved diagnostic methods given in books will fail us in a goodly number of cases. In carcinoma of the stomach loss of body-weight may be absent; it depends on the involvement of the pylorus. The craving for food may be normal. On the other hand, a sudden disinclination to take a particular variety of food, notwithstanding that it causes no distress, may appear early in the disease. Loss of strength is of greater diagnostic importance than loss of weight; often, it is not so much general weakness as disinclination to activity. A parallel condition may occur in the mind.—*Journ. A. M. A.*, vol. l, No. 23.

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# THE TREATMENT OF EARLY GONORRHŒA IN THE MALE.

By W. WYNDHAM POWELL, F.R.C.S.,

Formerly Surgical Registrar to the Westminster Hospital; Surgeon to the Westminster General Dispensary; and for many years Chief Clinical Assistant to St. Peter's Hospital, London.

It still seems to be a perennial question as to whether gonorrhœa should be treated locally with energy in the early stages, or more or less by the expectant method. I wish, therefore, to direct attention more especially to the abortive and irrigation treatment of early gonorrhœa while it affects only the anterior urethra, that is, the canal anterior to the compressor urethræ muscle, because it is while limited to this part that the most energetic measures should be taken to prevent its spread to the highly vulnerable and complicated region of the posterior urethra and its surrounding structures.

I hold that it should be a golden rule that if treatment is sought before the disease has invaded the membrano-prostatic area all the means in our power should be used to prevent it spreading there, and that this can be accomplished in the great majority of cases by the Janet method of irrigation with permanganate of potash is an undoubted fact.

If, then, a patient comes under treatment while the disease is limited to the anterior canal, the surgeon should be able to give a pretty definite promise that he will be able to control the inflammation and that its further spread can be prevented, provided that the patient can attend when required, and that in the acuter varieties his occupation does not necessitate excessive bodily movement. Not only can the spreading inflammation be checked but recovery of the part already affected is usually rapid and uneventful.

Gonorrhœa limited to the anterior urethra presents a totally different clinical picture, both as regards the constitutional effect on the patient and the disability it occasions, from that which presents itself when the inflammatory process involves the prostatic urethra. Again, ordinary occupations do not greatly influence the treatment of the disease by irrigation when confined to the anterior canal, whereas posterior (vesical) irrigations, if undertaken at all, require the patient to be at rest in any but the chronic stages.

The involvement of the posterior urethra in the inflammatory attack is not a complication, but is the natural course of almost all acute, especially first attacks. Subacute, especially second and subsequent attacks, often do not spread there.

"As a rule the infection spreads in between 80 and 90 per cent. of the cases through the entire length of the urethra" (Taylor).

"In 163 cases of acute and chronic gororrhœa, Jadassohn shows that in 87·7 per cent. the posterior was involved."

"Litzel's 53 cases of seven and ten weeks' duration, in only four the disease remained localised to the anterior urethra" (both quoted by Taylor).

"It may be assumed that in about 90 per cent. of all cases of gonorrhœa the posterior urethra is involved in the inflammation (White and Martin).

It behoves us, then, to encourage all patients to come for treatment in the early stages. This is the desirable period, and it should not be allowed to slip by without a determined effort being made to check it, and that its arrest is not difficult is well known to those who practise the irrigation treatment of this disease. It must not be forgotten that a first acute attack of gonorrhœa may spread to the posterior part with great rapidity, and that some infections, especially second and subsequent ones, may spread through the entire urethra without the subject being aware of the fact that he has an infection. It will be instructive to illustrate these varieties. A gentleman, æt. 27, came under my observation in October last. He had a first acute attack of gonorrhœa which began exactly sixty hours before. The infection took place four days prior to that. After washing out the anterior urethra very carefully with a pint of warm sterile water, by the method to be presently described, the urine was dull from the presence of mucus and some pus. The posterior urethra was already invaded. Being unable to keep at rest irrigations were not undertaken. He developed an acute prostatitis and was subsequently laid up for some weeks.

A medical man, whose previous (second) attack of gonorrhœa, by the way, six years ago, was aborted with silver nitrate on the second day of the discharge, came with a slight mucous secretion,

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slight heat on urination, and puffy meatus of twelve hours' duration. No intercourse for twelve days. His attention was first attracted to the part by the presence of a small pea-like nodule under the urethra near the frænum, which was an inflamed Littré's gland. He was under the impression that a small calculus had lodged there. After washing the anterior urethra thoroughly with a pint of warm sterile water some filaments in the urine showed invasion of the posterior urethra. He was not the subject of a chronic urethritis. No posterior irrigations were undertaken. Only after several examinations of the discharge could a few gonococci be found (Eastes). There were numbers of other micro-organisms. This was a double infection. He subsequently developed a prostatitis and the bacteriological cultivation and examination of the expressed prostatic discharge showed *B. coli* in pure culture. It is noteworthy that in this case, as in the next, urethroscopic examination showed that the glands of Littré were extensively affected, even extending into the bulb, and it is well known that this condition takes months to subside.

With regard to the infecting micro-organism no great distinction need be made in considering the suitability for abortive treatment. The non-gonococcal infections, while they do not usually occasion so acute an inflammation as the gonococcal are often more difficult to cure. This is why every precaution should be taken to prevent other microbes from entering the urethra in the wake of the gonococcus. In this connection the following case, which was a first attack, of non-specific urethritis, is well worth recording, as first attacks of this are rare:

A gentleman, æt. 25 years, who never had a previous urethritis, recently came under treatment on the fourth day of a slight discharge. Contact abroad seven days before the discharge appeared. Irregular irrigations kept the discharge in abeyance only. The posterior urethra was not invaded. On the seventeenth day a urethroscopic examination was made, and the glands of Littré were found to be extensively affected.

In this case the *Staphylococcus albus* was found in pure culture. Mr. Eastes, of the Laboratories of Pathology and Public Health, and Dr. R. W. Allen assisted me in the cultivation and the bacteriology of the discharge in these cases, and the latter refers to the last case in his book on opsonic treatment.*

No strong measures should be undertaken in these cases of affection of Littré's glands. The blandest of injections with an occasional anterior irrigation of weak permanganate is all that is necessary. Most of them recover perfectly in time, although they may take many months in doing so. It will be found that in the small percentage of cases which do not recover with irrigations, either the glands of Littré or the Morgagnian lacunæ have become affected at an early stage. A good example of the latter was in a man, æt. 40 years, with a first attack, where irrigations failed to cure, although they were successful in confining the disease to the anterior urethra. Urethroscopy on the thirtieth day showed one dropsical gland of Littré and several swollen pus-secreting lacunæ of Morgagni. Irrigations had no effect on these, nor had Kollmann's dilators gently applied and followed by irrigations. They were subsequently cured by a new method which I have devised, of inserting into each lacunæ a probe tipped with caustic in the air-distended urethra, under control of the eye. A description of this instrument, with the indications for its use, is about to be published. It is being made by Messrs. Down Bros., and is used with the aëro-urethroscope.

When a patient presents himself for treatment, after shortly going into the sexual history especially with reference to previous attacks of gonorrhœa and syphilis and their date, he is questioned as to when he had the exposure and as to the length of time that has elapsed since the very first sign of the disease. He should not have urinated for an hour or two. Observe if there is any evident discharge, if not gently press the terminal inch and see if any can be expressed. Microscopically examine any secretion, but the treatment need not necessarily be deferred for this. Having the irrigator ready with two pints of sterile water at a temperature of 98° F. and at the usual height of five or six feet from the penis, wash out the terminal two inches of the urethra with half a pint, the patient compressing the penis at the proper spot. Wash out now the next two or three inches, the organ being grasped by the patient with the thumb and one finger behind the scrotum and as

* 'The Opsonic Method of Treatment,' H. K. Lewis, 1907.

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near to the pubic arch as possible. After this, wash out the bulb by the ordinary method of an anterior irrigation (*q.v.*) with a gentle pressure at first to avoid forcing any fluid and discharge into the posterior urethra, for this would vitiate your diagnosis. While increasing the pressure and ballooning the urethra allow the latter to empty itself completely by removing the irrigator five or six times during the flushing-out process. The circulation of the fluid in the far end of the bulb is very imperfect, and it is astonishing what care is necessary to remove all the shreds of discharge from it when affected. Do it thoroughly, as it is a most critical proceeding bearing on the diagnosis and treatment. Use no catheter, backflow or otherwise, as nothing equals a proper flush out from the meatus with an irrigator.

Now let the patient urinate into a clean glass free from bits of cotton. The presence or absence of filaments in these washings and the clearness and freedom from shreds of the urine give roughly the necessary information as to the extent of the disease. I say roughly, because in the initial stages of the inflammation only a slight cloudiness may be observed, and this may be easily missed. If there are visible phosphates in the urine add acid to clear, but do not be deceived by any milkiness that the acid produces from the presence of the products of sandal oil, copaiba, etc., in the urine. One should be satisfied that the case is not a relapsed gonorrhœa, for these patients often come with the idea that a fresh infection has occurred. These generally show filaments in all the washings and often in the urine also, and the discharge is apt to appear the day following the intercourse. One irrigation will often suffice to apparently cure these cases, but the latent disease remains.

Under no circumstances do I trust to the two-glass test. It is a most unscientific method. Posterior invasion can be diagnosed perhaps twenty-four hours earlier by the wash-out test than by the two-glass. This is of the utmost importance in cases where the patient is prepared to have posterior irrigation in the event of this part becoming affected. There need be no fear of untoward results if posterior irrigations are undertaken in the initial stage of the inflammation of this part, provided that the patient can keep at rest and that a little cocaine is used to prevent spasm and injurious pressure. Again, in slight chronic superficial affections of the posterior urethra the first flush of urine removes with it all the filaments, as anyone can prove for himself by performing an anterior wash-out and having the urine passed into two glasses. The first glass may contain all the shreds.

*The abortive method.*—Assuming, then, that the patient comes within a few hours of the initial symptoms, after the washing-out process has been gone through, and you are satisfied there is nothing beyond two inches, inject 1 per cent. silver nitrate solution into the terminal one and a half or two inches of the urethra—(an eye dropper is a convenient instrument)—letting this out and repeating the process six or eight times and taking one or two minutes over it. The patient grips the penis at the proper place to prevent the solution going further. It is well, also, to "manipulate" thoroughly, but not roughly, the urethra from the outside while holding the solution in to assist its penetration into any duct or lacuna. This is why this method should be adopted in preference to the application of silver through the urethroscopic tube. If the disease were more than a few hours old then it depends on the acuteness, as shown by redness, œdema, and irritation, whether the silver process should be undertaken or not. In first acute attacks after twelve hours, and always if in doubt, the silver should be omitted. If quite subacute, especially if not a first attack, then silver may be applied up to thirty-six or even forty-eight hours from the beginning of the symptoms, provided there are no shreds beyond the part intended for treatment. In these cases two to two and a half inches of the urethra may be treated with a 1 or 1½ per cent. silver nitrate solution, and immediately followed by an irrigation of the whole anterior urethra with two pints of a 1 in 2000 permanganate solution. Rest should be enjoined after this and urination deferred for three or four hours. Twelve hours or so after this treatment permanganate irrigations should be commenced, and any discharge carefully examined to ascertain whether or not the silver process has been successful, because, if not, irrigations must be continued systematically until no more gonococci are found. It is not unusual for one strong permanganate irrigation to clear up the case where gonococci have been found after the silver process, and on the

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other hand many irrigations may be necessary. Ten drops of a 4 per cent. β-eucaine lactate solution may be used to anæsthetise the anterior part of the penile urethra (cocaine hydrochlorate precipitates silver salts, forming an insoluble chloride). Never apply the silver nitrate solution to the whole anterior (bulbo-penile) urethra. If it fails to arrest the disease it undoubtedly favours its spread backwards.

*Irrigation by the Janet method.*—This is done with permanganate of potash solution of varying strength, and the force necessary to flush out the urethra is obtained by raising the reservoir to a certain height, usually 5 or 6 feet from the penis. The tension in the urethra is obtained by rhythmically obstructing the outflow therefrom with the finger and thumb of the left hand as will be explained later. The strength of the solution varies between 1 in 1000 and 1 in 5000 for anterior irrigations, and the quantity 3 pints. My favourite formula is 3 pints of 1 in 1500 at a temperature of 105° to 107° F., and a height of 6 feet for a subacute case and administered once daily. For a very acute case half this strength given twice daily for 3 or 4 days, then the stronger solution once daily. Owing to the varying behaviour of different urethræ and the idiosyncrasy of the patient it is impossible to lay down definite rules for the guidance of the surgeon. The best that can be done is to generalise and to leave some of the details to his judgment. An "attack of gonorrhœa" conveys the idea to some that there is one formula to meet it. Nothing can be further from the truth. Pure routine without the proper appreciation of small signs and symptoms will lead to but indifferent success. Let the beginner therefore undertake only subacute or chronic cases until he is sufficiently familiar with the details of irrigation to undertake a hyper-acute case. I sometimes have coal "suitable for all purposes," but in the kitchener it burns furiously while in my dog-grate it burns feebly enough. Equally variable will be the effect of your favourite 1 in 1500 solution of permanganate in different urethræ. I will therefore not be tempted to give a set table of strengths, but merely state what my practice is for anterior irrigation.

(1) Hyper-acute cases, 1 in 2000 to 1 in 4000 twice daily for 3 or 4 days, then once daily with 1 in 1500 to 1 in 2000.

(2) Subacute cases, 1 in 2000 to 1 in 3000 twice daily for 3 or 4 days, then once daily with 1 in 1000 to 1 in 1500.

(3) Painless cases, 1 in 1000 to 1 in 1500 daily.

Pain and œdema are the chief indications for reducing the strength. In acute cases pain after an irrigation is sometimes considerable, but should not be more than a slight smart at the first urination, which is always deferred for 3 or 4 hours after the irrigation. If pain is excessive reduce the strength. Again, where strong solutions are used owing to great anxiety on the part of the patient for a rapid recovery the serous discharge may rarely become blood-stained. Reduce the strength at once or use boric acid solution until this disappears.

Do not use a strong cocaine solution in these cases, because absorption may be rapid. Ten to 20 drops of a 1 per cent. cocaine hydrochlorate solution or 4 per cent. β-eucaine lactate held in for a minute or two would tide over the actual irrigation. Do not anæsthetise the region of the bulb, as this facilitates the passage of the fluid into the bladder. Always treat a highly inflamed and tender urethra with the greatest possible gentleness. Any traumatism beyond what is actually required for its proper flushing out will, without doubt, delay recovery. This is why the closing of the meatus with the finger and thumb to obtain the tension in the urethra is to be preferred to blocking it with the nozzle. After an anterior irrigation ask the patient to urinate if he has any distinct desire to do so in case some of the solution may have entered the bladder.

If the patient is able to pay only irregular visits for irrigation he may also use the permanganate thoroughly three or four times daily with a hand syringe.

In cases where the inflammation has spread to the posterior urethra and complete rest is out of the question, irrigate the anterior occasionally with quite a weak solution in case it enters the posterior part and irritates it.

Do not hesitate to cease irrigations abruptly The gonococcal infection may terminate very suddenly (unlike the other infections). Other micro-organisms (except in double primary infections) do not get a footing in a urethra that is under active irrigation treatment. Request the patient to come if the least opaque discharge shows itself

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the first thing in the morning. Discourage the patient from constantly stripping the organ to look for discharge. Give some cover glasses with directions how to place a little of the discharge on them should any show. The microscope must be in constant use in these cases. When recovery is doubtful the patient's visit should be in the early morning before urinating. A persistent bead of pus in the morning, occurring during irrigations, is highly suggestive of an affection of the lacunæ, or of some minute canal, or of suppuration of a Littré's gland. It should be borne in mind that some cases of gonorrhœa are very easily cured by almost any form of local treatment, and especially by irrigations, and no general conclusions should be drawn from single cases. The epithelium of a urethra that has suffered from a prolonged attack or from many attacks becomes changed and hardened, and is less easily penetrated by the gonococcus. In fact in old cases where much fibrous sclerosis has taken place the columnar epithelium is converted into the squamous variety.

A gentleman, æt. 26 years, who had been under my care two years before for a prolonged gonorrhœa and who had recovered completely, came, two or three weeks ago, having a free, painless discharge of five days' duration. Intercourse up to the day of the discharge. The specimen was crammed with gonococci. Many heavy flakes were washed out even from the bulb. There was nothing from the posterior urethra. One irrigation of three pints of 1 in 1500 permanganate at a temperature of 110° F. and a height of six feet cured the case. As the irrigation was painless the urethra was "ballooned" as much as possible and the solution used warmer than usual. There were subsequently no filaments in the morning urine, and cohabitation and alcohol were resumed a few days afterwards. Immunity may play a part in these cases.

The most noticeable feature in cases that come under treatment after having passed through the hands of the family practitioner, is the fact that the least deviation from the ordinary manner of infection or the unclassical way in which the symptoms developed is sufficient to cause him to pronounce the case non-gonococcal. This happens with such astonishing frequency that it is well to direct attention to it. Most of us have seen cases where it has required a month's cohabitation or more to infect the male urethra. That the gonococcus (? or that particular strain) may become almost inert to the particular urethra or female genital organs in which it has long been resident is well known. This is sometimes seen in husband and wife who are apparently quite well, but the interloper finds out to his sorrow how deceptive appearances may be!

It is exceedingly difficult to find the gonococcus in the female in old cases. It is almost useless to request the infecting woman to consult her medical adviser with the object of throwing light on the nature of the infection conveyed. The answer is almost always the same—that the gonococcus cannot be found. This diplococcus probably descends from the upper reaches only during the menstrual flow, which leads to further confusion, the surgeon saddling the ordinary discharge with the cause of the infection instead of the gonococcus.

A gentleman left his mistress in charge of another man while he was away. This man contracted a first gonorrhœa. As all three were astonished, the woman was examined by three eminent specialists. All said she was free from gonorrhœa. I invited the first man to see me, and while protesting that he was quite well he had many filaments in his urine, and a milky discharge could be expressed from his urethra. Although this case is not conclusive evidence it is highly suggestive of a residual gonorrhœa in the man and woman.

I think that gynæcologists are more to blame than the genito-urinary surgeon for the prevalence of gonorrhœa. It is true that they receive no assistance from women in the matter. Women are so accustomed to discharges that they will harbour for years an unsuspected gonorrhœa. They are always highly indignant if such a possibility is suggested, even when you know that infection has been conveyed.

The incubation period is another pitfall in the question of diagnosis. I have known it as brief as thirty-six hours in a first case. On the other hand, I have known it to be prolonged for a month in a first case. A fortnight is by no means unusual. The following case is one of twenty days' duration: An officer who had recently returned from the East Coast of India came to me with a free painless discharge of six weeks' duration. The specimen showed countless gonococci (Allen). This was his first attack. On January 30th he dismissed his mistress and went up country, constantly riding

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and taking active exercise, and taking a little alcohol in the usual way. On February 19th, two days before his return, a stickiness with a little painless discharge appeared at the meatus. There had been no intercourse since January 30th. The surgeon thought the case could not possibly be one of gonorrhœa.

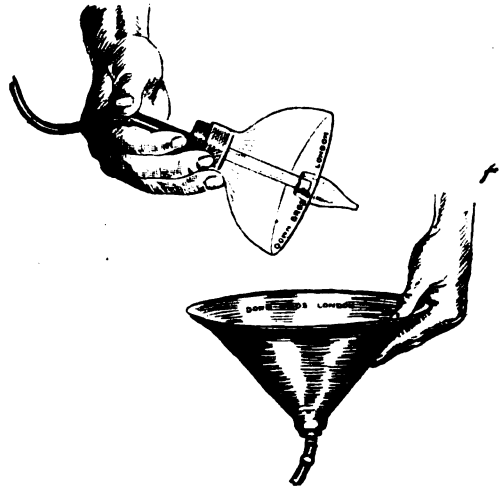
It is of the utmost importance to grasp thoroughly the theory of irrigation as well as to pay the greatest attention to the few small details necessary for its successful practice. Although very simple and easily explained yet some essential feature must have been missed by those who have tried the method and found it wanting.

*Technique of irrigation.*—The irrigator which goes under my name is very simple and is the one used at St. Peter's Hospital. It consists of a piece of barometer glass tubing about five inches long which passes through a rubber cork, which in turn plugs into a glass cup or shield which catches the spurting fluid on its return from the urethra. The glass nozzle for the meatus fits on to one end of the glass stem and the rubber tubing from the reservoir on to the other end. It can easily be taken to pieces for boiling. The apparatus rests, during an irrigation, on and between the tips of the third and fourth fingers of the right hand, while the outer end of the glass stem is grasped between the thumb and the middle of the forefinger. Reference to the illustration will explain this better than words.

The thumb and forefinger not only steady the apparatus but also control the flow by compressing the rubber tube partially or completely as may be required. This is exercised only at the beginning of an irrigation, when gentleness is necessary and when the glans is being cleansed, or when the stream is stopped altogether, otherwise no compression of the tube is necessary. The tension in the urethra (ballooning) is obtained, not by altering the height of the reservoir, but by partially or completely obstructing the return flow from the urethra, by compressing the meatus rhythmically with the forefinger and thumb of the left hand above or below the point of the nozzle once, twice, or three times to the second according to the capacity of the canal and the size of the bore in the nozzle. An irrigation, therefore, consists in a rapid filling and emptying of the urethra.

The glass nozzle is detachable and should have a bore which admits a No. 9 F bougie. An unsatisfactory flush is obtained with anything smaller. The operator stands on the right and a little in front of the patient. The penis is steadied between the middle and ring fingers of the left hand. This leaves the thumb and forefinger free to regulate the outflow. The inflow must be in the proper axis of the urethra. With the patient standing up or sitting on the edge of a chair, the direction must be slightly downwards as well as backwards, otherwise the stream will impinge on the roof of the canal and cause much soreness at one spot.

It is well also to move occasionally the tip of the glass nozzle from the lower sulcus of the meatus to the upper, the meatus being compressed by the finger and thumb above or below as the case



may be. The nozzle must be removed for boiling after each case.

The reservoir should be of three pints' capacity and raised five or six feet from the penis for anterior, and four or five feet for posterior irrigations. The clip on the tube from the reservoir should be such as can be manipulated easily with one hand. A large funnel with rubber tube and held by the patient catches and conducts the waste fluid away. Water should be run through the apparatus immediately after use. A strong solution of oxalic acid will remove all permanganate stains.

As to the local action of the permanganate it is difficult to explain although easy to theorise. Its effect is to a great extent mechanical, and with the stronger solutions slightly caustic also (which is

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a phase of the mechanical). The mucous membrane is stained and rendered dry, dull, and inelastic, as I have often verified with the urethroscope. It is thus converted into an unsuitable soil for the growth of the gonococcus. Probably excess of toxins formed on or near the surface interferes with phagocytosis more than does the chemical. In any case any gonococci under the mucous membrane appears to be very easily disposed of. But the gonococcus clings very closely to the mucous membrane itself and its lacunæ, and if it extends deeper is often associated with one of the more deeply placed glands of Littré.

I have never had evidence of the entrance of the gonococcus or its toxins into the general circulation during anterior irrigations (the posterior not being involved). Frequently previous attacks have been followed by rheumatism, which does not recur during subsequent infections if irrigated. One of my series of twenty cases had a bad attack of iritis and rheumatism, which laid him up for six weeks during his previous gonorrhœa two years before. No relapse followed the present attack, which was confined to the anterior canal by irrigations.

A patient who was recently under me for a second gonorrhœa which had already involved the posterior, had a mild double conjunctivitis with a little muco-purulent discharge in the corner of each eye. No gonococci could be found in this discharge. He had just such an attack with his first gonorrhœa. It looks as if the conjunctiva was assisting the urethral mucous membrane to get rid of the toxins.

A short analysis of the last twenty consecutive cases of fresh urethritis in private practice, who came in the early stages while the infection was limited to the penile or bulbo-penile urethra, will be sufficient to illustrate the influence of irrigations in controlling the spread of the disease.

*In nineteen cases the inflammation did not reach the membrano-prostatic urethra.* In one the disease spread to this part from interruption of treatment owing to holidays.

Of these cases six were first attacks.

Six were acute, nine subacute, and five were painless.

The incubation period varied between two and nine days. At the first visit the discharge had been in evidence from twelve hours to five days.

The nitrate of silver process was adopted in seven of the cases.

Six were aborted in one day. Five others recovered within one week. The longest was twenty-four days in recovering. This case, which was sent to me by Janet, of Paris, had already received a couple of irrigations before I saw him. He was probably cured in eighteen days, but, as is often the case, at the patient's request there are several unnecessary irrigations at the end "to make sure" that recovery is complete.

One became chronic and was subsequently cured by means of the urethroscope. One left for India uncured (the non-gonococcal case before mentioned). In one case, which was probably non-specific, a little serous discharge persisted owing to a phosphaturia. One case of gonorrhœa was peculiar in that it was probably contracted from the seat of a water closet—a medium usually reserved for the members of a certain calling. He had a mild attack thirty years before which recovered perfectly. His mistress left town on business on October 10th. Seven weeks after, on November 28th, a gonorrhœa started. It appears that the valet, whom I saw, had an acute first attack and used the same closet. The man had no doubt himself that this was the source of his infection. He recovered rapidly and had only nine irrigations in all.

When no more secretion can be expressed from the terminal inch of the urethra on rising in the morning, and the meatus is not glued, and there are no filaments in the urine, the case can be regarded, tentatively, as having recovered. Careful observation must, however, be kept up for another week or so, as any injurious influences which tend to weaken the resisting power of the urethral mucosa, such as alcohol, excessive exercise or intercourse, may enable any semi-moribund gonococci that happen to be lurking in the recesses to take on a new lease of life. This is exactly what occurred in the last case. Alcohol and a long walk in the evening, which was done expressly to test the cure, caused a minute bead of pus, containing gonococci, to appear in the morning, two or three days after apparent complete recovery. One strong irrigation, however, finally disposed of the disease.

In a subject of chronic filaments in the urine it is not so easy to say when recovery from a fresh attack takes place. The microscope will be of great assistance, and the urethroscope will be of service if the surgeon is thoroughly accustomed to its use.

*June 22nd*, 1908.

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# THE VERMIFORM APPENDIX AS A PELVIC ORGAN.

A Clinical Lecture delivered at the Middlesex Hospital.

By J. BLAND-SUTTON,
Surgeon to the Hospital.

THE position occupied by the tip of the vermiform appendix is only limited by the length of this tubular organ. I have often found the tip of the appendix in contact with the gall-bladder when the latter has been normal in size and in position, but I have more often seen the end of the vermiform appendix lying in the pelvis and resting on the rectum. As a pelvic organ the vermiform appendix is as liable to infection and perforation as in any other position; in such circumstances, especially in women, it is responsible for many errors in diagnosis. An acute appendicitis when the tip of the appendix is free in the pelvis mimics, not only pelvic abscess, tubal pregnancy, and pyosalpinx, but it may actually set up acute salpingitis and ovaritis. It is a fact that subacute and chronic diseases of the appendix may extend to, and involve the right ovary and Fallopian tube; it is of importance to remember that in acute and chronic affections of the right half of the female pelvis the ovary, tube, and appendix are in such close proximity that it is difficult and often unwise to make a positive diagnosis between primary inflammatory disease of these three organs.

When attention was first drawn to the frequency of appendicitis many statements were made as to its infrequency in women and girls. I published a paper on "Appendicitis in Girls and Women" ('CLINICAL JOURNAL,' 1900, vol. xv, p. 321), to show that this opinion was without foundation, and subsequent experience has amply confirmed it.

In this lecture I propose to bring under your notice some cases of more than usual interest, in

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which great difficulties in diagnosis were set up in consequence of the propinquity of the right ovary and Fallopian tube to the vermiform appendix. One of the most remarkable examples which has come under my notice I reported two years ago. The patient had signs and symptoms indicating acute septic salpingitis, such as we so often see as a sequel to gonorrhœa, but she was living in circumstances which made it difficult to believe in the diagnosis. At the operation I found the appendix adherent to the cœlomic ostium of a distended right Fallopian tube. The parts were carefully removed wtthout disturbing their relationship. After hardening, the parts were carefully bisected, and it was then easily demonstrated that the girl had acute appendicitis; perforation had occurred near the tip of the appendix, which had become surrounded by, and adherent to, the tubal fimbriæ, and the inflammatory and infective stuff from the

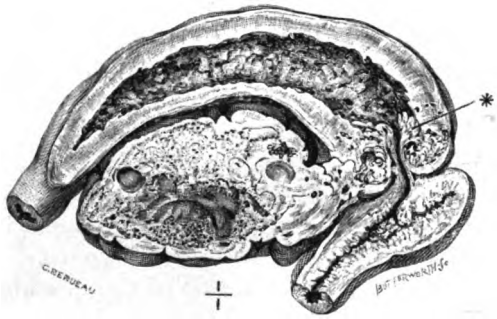

FIG. 1.—An ovary with the Fallopian tube and vermiform appendix shown in section. The tip of the appendix is perforated and the pus was discharged into the cœlomic ostium of the tube and produced salpingitis.

appendix had discharged itself directly into the lumen of the Fallopian tube, causing an acute salpingitis (Fig. 1). The patient recovered.

The drawing illustrating this case is worth attentive examination, because the fimbriæ are curled inwards and tucked into the lumen of the tube exactly as happens when the cœlomic ostium is occluded as a sequel to acute salpingitis (salpingitic occlusion). When this ostium is occluded as a consequence of inflammation of adjacent tissues the fimbriæ are merely matted together.

I am able to show you an example of this: A girl, æt. 11 years, complained of pain in the right flank accompanied by the usual signs of appendicitis; the urgent signs subsided but pain persisted in the right iliac fossa, which made it judicious to remove the appendix. At the operation the tip of the appendix was found adherent to the right ovary and broad ligament: from these structures it could be easily detached but the inflammation had so matted together the tubal fimbriæ as to occlude the cœlomic ostium of the tube (perimetric occlusion). I removed the outer third of the tube but succeeded in conserving the ovary. In this case the trouble depended on a simple inflammation of the appendix spreading to the ovary and tube, but when it happens to be due to infection of a virulent type matters do not end so favourably. Here is a set of specimens consisting of a sloughing appendix containing fæcal concretions, a Fallopian tube and the corresponding ovary also in the condition of

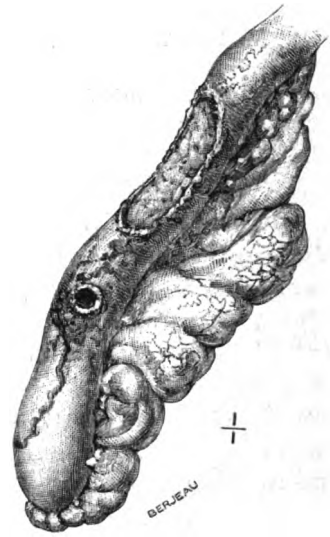

FIG. 2.—A vermiform appendix with a perforation near the tip.

slough. These parts were removed from a woman, æt. 35 years, attacked with fulminating appendicitis. She owes her life to a prompt operation. The hole in the appendix indicates that the disease began in this organ and spread to the ovary and tube.

On one occasion whilst operating on a young man for an acute appendix abscess I felt a smooth rounded body in the abscess cavity; it felt like a testis, so I at once examined the scrotum and found that it only contained one testis, the left. His father was in an adjoining room, so I explained the situation to him and obtained his consent to remove the retained testis.

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A good deal of attention has been devoted of late to the effects of inflammation spreading from the appendix to the right tube and ovary. Some of the more important papers have been written by Arnold Lea, Handley, and Hartmann.

Although the vermiform appendix when inflamed may perforate at any point, yet in the greater number of instances it occurs at or near its free end. Those who rely on the gross anatomy of this organ attribute the vulnerability of the tip to the fact that it is supplied by a terminal artery. In 1891 I endeavoured to show that the structure of this part of the appendix might account for it. The tip of the vermiform appendix is rich in adenoid tissue, like the tonsil (Fig. 3); this tissue

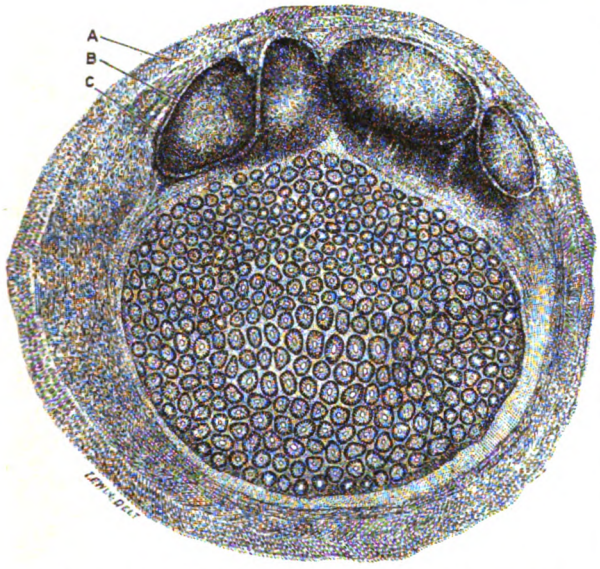


Fig. 3.—The histological arrangement of tissues at the "tip" of the vermiform appendix. A. Thickened serous coat. B. and C. Clump of adenoid tissue.

is notoriously unstable and peculiarly liable to infection. Indeed, the terminal portion of the appendix may be regarded an an abdominal tonsil. Septic infection of the adenoid tissue in the tonsil when it leads to quinsy bursts into the patient's throat and ends happily, save in the rarest cases, but the bursting of an appendix is always a serious matter, and often a catastrophe.

The most important occasion on which to keep in mind that the vermiform appendix is occasionally a pelvic organ, is when it perforates and discharges infective material directly into the pelvic cavity, converting it into a pool of stinking pus. This happens in men and women and gives rise to very acute and urgent symptoms. I find it necessary to emphasise this, because when called into consultation on such deadly conditions it is so difficult to persuade practitioners that the illness is due to a perforated appendix, in the absence of pain, or swelling, over the cæcum. Another point they urge against immediate operation is the absence of rigors. In the first paper I wrote on this subject (see 'Transactions of the Clinical Society,' 1901, vol. xxiv, p. 116), it was pointed out that the presence of abominably fœtid fæcal-contaminated pus in large quantity due to perforation of the appendix is not indicated in the usual orthodox manner by rigors. Eighteen years' subsequent observation and an experience of many hundred operations for appendix disease of all kinds has confirmed this early opinion; I have never seen rigors associated with appendicitis.

Other points I wish to impress upon you very strongly in regard to these acute and subacute pelvic abscesses depending upon perforative lesions of an intra-pelvic appendix. When you are called upon to deal with them bear in mind three things:

(1) Operate immediately you have satisfied yourself of the nature of the case.

(2) Make your incision in the middle line, not only because the classical "median subumbilical incision" affords the best access to the pelvic cavity and the appendix, but it is the direct route for drainage.

(3) Be sure and remove the appendix.

The operation to be effectual should be done in ten or fifteen minutes: don't fiddle about with any fancy method of ligaturing the stump, but tie it and its mesentery with a single piece of silk and introduce a big drainage-tube and get the patient off the table as quickly as possible. Some of you have seen me do this many times and can bear witness to the good consequences. Intra-pelvic collections of pus in boys and girls under twelve years of age the consequence of appendix perforations are very fatal, because at this age the great omentum is a scanty organ, and is not able to confine the pus in the pelvis as happens in later life. Many men and women owe their lives to this beneficent action of the great omentum.

I wish to particularly urge on you the importance of removing the vermiform appendix in such operations, not only because it saves the patient

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from the anxiety, the risk, and expense of a second operation, but because it shortens the convalescence and saves him the annoyance of a troublesome sinus. It is necessary to emphasise this matter, because, judging from the great number of patients which have come under my notice during the last three years, in whom I have had to remove an appendix left by other surgeons, I fear it is becoming a prevalent practice to merely drain an acute appendix abscess and then remove the vermiform appendix some weeks later. To my mind this is pernicious teaching. Those of you who witnessed the difficulties which were encountered in some of these operations may take the lesson well to heart, and make it a rule to do your best and remove the swollen and easily detachable appendix in the acute stage.

Once, and once only, have I failed to find a vermiform appendix. The patient, a girl, æt. 15

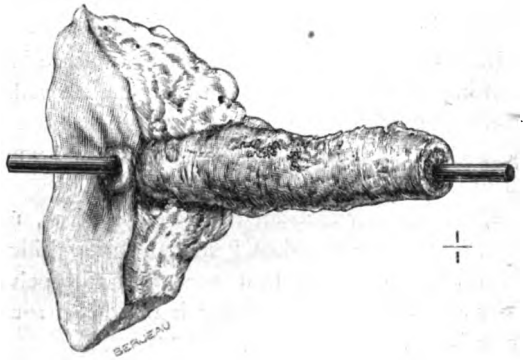

FIG. 4.—A vermiform appendix with its perforated tip forming a sinus discharging on the scar of a cœliotomy incision.

years, was under the observation of an astute physician who recognised that her illness was due to pelvic inflammation, but considered it to be pelvic cellulitis. I believed it to be a collection of pus depending on the perforation of an intra-pelvic vermiform appendix. The girl was very ill; I let the pus out by means of a median sub-umbilical incision and endeavoured to find the appendix. The patient being too dangerously ill to permit of a thorough search a big drain-tube was introduced and she was returned to bed. To my astonishment and pleasure this girl recovered, but with a tiny sinus. This surprised me as nothing was ligatured. A year later, on the urgent representation of the doctor who had charge of the patient in the country, I agreed to deal with the sinus. I dropped a probe down the sinus and then proceeded to cut round it. You may judge of my surprise on finding that the cutaneous orifice of the sinus represented a hole in the tip of the vermiform appendix (Fig. 4), and that when a probe was introduced into this little opening it traversed the canal of the appendix and went into the cæcum. There were no adhesions and the operation resolved itself into a simple appendectomy which ended successfully.

I have recently had a patient under my care which illustrates in a striking way the importance of ascertaining the condition of the vermiform appendix when operating for a supposed appendix abscess. A nursemaid, æt. 22 years, underwent an operation for what was regarded as an acute appendix abscess in a provincial hospital. The pus was evacuated and a drain-tube introduced; in a few days a fæcal fistula formed and persisted for two years and three months. During the whole of this period she was confined to bed. She came under my care at the end of this time with the hope of having the fistula closed. I opened the abdomen by means of a median subumbilical incision and found the vermiform appendix normal. There was bilateral disease of the tubes and ovaries; these were removed. A hole was found in the sigmoid flexure, necessitating resection of three inches of this gut and circular enterorrhaphy; and a hole in the ileum required removal of a foot of this part of the intestine and end-to-end junction. She recovered. Her original trouble was a pelvic abscess, secondary to tubal disease (salpingitis).

### *On Pins in the Vermiform Appendix.*

It would, perhaps, scarcely occur to you that when a pin finds its way into the appendix, the effects which this accident may occasion are modified according to the position of the appendix, and may be accentuated if it be a pelvic organ. I have had occasion to look into this matter lately, mainly on account of a case which came under my observation.

A spinster, æt 24 years, had a sudden attack of severe pain in the hypogastric region which caused her to take to bed. Gradually the pain subsided, but did not disappear. Six weeks later as the patient's condition did not improve, I was asked to see her by Dr. Cowan. The signs

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were those of subacute appendicitis, and nothing abnormal could be detected in the pelvis. A few days later I explored the appendix region through the usual lateral incision, and found a long vermiform appendix reaching into the pelvis, but immediately below the line of the iliac vessels it firmly adhered to the pelvic wall, and at this point the appendix was swollen and appeared to contain something extremely hard. I at once suspected that it contained a foreign body, and took care to remove the thing entire.

The appendix when removed measured five inches, and the swollen portion contained two stony concretions. When these came to be examined a pin was found as a nucleus. If this patient had been skiagraphed, the calculus would have produced a strong shadow on the plate and led the surgeon to believe that he had to deal with a stone in the ureter (Fig. 5). I had never seen a concretion of this kind in an appendix previously, but the similarity in appearance, the mode of formation, and

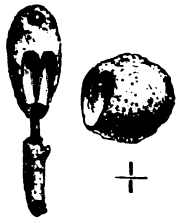

FIG. 5.—A pin from the vermiform appendix encrusted with calcium phosphate.

the situation in which they occurred at once reminded me of the colic calculi so common in horses. I was fortunately able to enlist the interest of Mr. A. M. Kellas, B.Sc., Ph.D., in this matter, and he made an analytical examination of this appendix-concretion and compared it with the composition of an equine colic calculus, of which we have many in the museum. These concretions in horses form around foreign bodies, especially scraps of metal, and are common in the cæcum. Sometimes they are as big as a man's head. I was amused some years ago on visiting a veterinary museum to find a big colic calculus doing duty as a door stop! A typical calculus is represented in section, and this is the specimen analysed by Mr. Kellas; he reports that "The calculus consists essentially of ammonium magnesium phosphate. A trace of iron is present."

The concretion around the pin from the appendix "consists essentially of calcium phosphate with a small quantity of calcium oxalate, about 6 per cent. of fatty material; a large trace of iron (perhaps as phosphate), and a trace of magnesium ammonium phosphate are present."

A great many cases have been reported in which a pin has been found in the vermiform appendix in the course of an operation for appendicitis, and in one case two were present. Kelly has collected forty-six reported cases, and it is of interest to note that it is quite exceptional for the patients to have any knowledge that they swallowed pins. This is equally true of pins found in connection with anal abscesses.

When a pin creates a disturbance in the vermiform appendix and this structure hangs in the

FIG. 6.—A calculus from the cæcum of a horse divided to show the nucleus, which is a fragment of metal.

pelvis, the pin, when it pricks its way through the walls of the appendix, occasionally punctures the bladder. In a remarkable case recorded by Kingdon in 1842, a boy, æt. 7 years, used to pass worms from the urethra. When he died the appendix was adherent to, and opened into the upper part of, the bladder; a calculus occupied the bladder with a pin as its nucleus, and it was believed that this pin had reached this situation by way of the appendix. Keen, in the course of an operation on a man, æt. 24 years, found a long appendix with its tip firmly adherent to, and discharging fæces into, the bladder. The history of this case, which is of great interest, suggested

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that a pin, which the patient had swallowed, and which had been removed from his urethra when he was seven years of age, may have caused the trouble.

Pelvic abscesses arising from perforation of the vermiform appendix occasionally open into the bladder independently of pins.

The vermiform appendix occasionally occupies a hernial sac. A case is known in which the appendix with a pin protruding through its wall has been found in the sac of an inguinal hernia (Roberts).

In the past a number of human beings have lost their lives (and many more have suffered a long and serious illness) in consequence of a pin accidentally swallowed, pricking its way through the wall of the vermiform appendix. Thus these pins actually fill the part described in the speech which Shakespeare puts into the mouth of Richard II:

"For within the hollow crown
That rounds the mortal temples of a king
Keeps death his court; and there the antic sits,
Scoffing at his state, and grinning at his pomp,—
Allowing him a breath a little scene
To monarchize, be fear'd, and kill with looks;
Infusing him with self and vain conceits,—
As if this flesh which walls about our life,
Were brass impregnable,—and, humour'd thus,
Comes at the last, and with a little pin
Bores through his castle walls, and—farewell, king!"
(*Act II, sc.* 1).

*June 29th*, 1908.

**Internal Secretion of the Ovary.**—M. Hughes Alamartine, after a careful consideration of the evidence for the theory that there is an internal secretion of the ovary of importance to the body, states that it is his conviction that this fact is established by the evidence we now possess. There is a correlation between this gland and the supra-renal capsules and other glands which have an internal secretion. There are formed in the ovaries toxic substances, and this goes to show that the action of this organ is to remove from the body substances that are unfavourable to its action, and which are brought to the ovaries by the circulation. The two functions, antitoxic and sexual, need not be separated.—*Med. Rec.*, vol. lxxiii, No. 23.

# SOME INTER-RELATIONS OF ABDOMINAL AND THORACIC DISEASES.*

By SIR WILLIAM HENRY ALLCHIN, M.D., F.R.C.P.,
Consulting Physician to the Westminster Hospital and to the Victoria Hospital for Children.

(*Concluded from page* 166.)

HAVING thus before us the main features of the anatomical mechanism through which are affected such relations as may be found to exist between disease as it affects the thorax and the abdomen, we are in a position to consider these morbid states somewhat in detail, more especially with a view to their full pathological comprehension and their clinical differentiation. For there can be no doubt that some extremely difficult problems present themselves at the bedside in connection with abnormal conditions in the neighbourhood of the diaphragm, in which the application of physical methods of investigation are not so completely applicable as elsewhere, and where the results of inquiry are more doubtful. That some very abnormal state is existent may be confidently affirmed, but neither its exact nature, nor what from a treatment point of view is equally important, its exact location, are by any means clear; and even should these points appear in some measure to be probable, there remains the starting-point of the mischief to be determined—no unimportant matter either for prognosis or management.

Considering the extreme tenuity of the structures that separate the serous linings of the several cavities of the chest and abdomen, and the close connection of the lymphatics draining these sacs, it might be expected that inflammatory conditions originating in one would readily spread to the others. This is not so, however; as a matter of fact the extension is comparatively rare, and certainly so to an extent that gives rise to recognisable indications. This may be compared to the infrequency with which a pleurisy of one side involves either the other side or the pericardium, or that a pericarditis sets up a like affection in either pleura.

* Introductory Address delivered before the Post-Graduate Course, Mount Vernon Hospital for Diseases of the Chest.

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It is also worth noting in passing that acute pleurisy or acute pericarditis are singularly uncomplicated affections, they seldom involve to any very appreciable degree the viscera which the serous membranes cover, and whilst a pneumonia may lead to a pleurisy, the spread in the reverse direction is almost unknown,* though a simultaneous involvement of lung and serosa is of course common. A pleurisy sometimes accompanied by pneumonia is practically the only complication of acute peritonitis, and is estimated to occur in only 20 per cent. of the cases, and this is said to be more frequently present on the right side.† I am not prepared with figures to show the proportion of cases of acute peritonitis which develop secondarily to a pleurisy or pericarditis, but I believe I should be right in saying that such a complication of either of these affections is, to say the least, very uncommon, considering the frequency with which they are met with. But the possibility of a pleurisy secondary to peritonitis, and to a much less degree the reverse occurrence, is not to be lost sight of, since the secondary affection may be but slight and yet sufficient to maintain general symptoms, such as fever, for which the original malady may improperly get the credit. Together with this mutual implication of the thoracic and abdominal serous membranes one is insensibly led to remember the striking difference exhibited by these structures in their response to certain infections, more especially the rheumatic, for whereas the pericardium, and to a lesser degree the pleura, are prone to manifest considerable inflammatory changes in response to the invasion of the rheumatic infection, a peritonitis from the same cause is of extremely doubtful occurrence.

In Bright's disease, on the other hand, peritonitis of an acute or subacute type, and purulent in character, is a terminal affection in about 10 per cent. of the cases, and the statistics on the subject, prepared by the late Dr. Sibson,‡ showed that among the cases of Bright's disease collected from various sources 8·1 per cent. were attacked by pericarditis, whilst pleurisy occurred twice as frequently.

There remains to be mentioned in this connection a somewhat rare form of disease to which attention has been specially directed within recent years, more particularly with regard to the relation of certain forms of perihepatitis and perisplenitis to chronic peritonitis. The disease in question has received several names, but that suggested by Dr. Nicholls,* of Montreal—"multiple progressive hyaloserositis"—appears to be the most suitable, although polyserositis, except for its hybrid derivation, might be preferred. "In its complete development it is characterised by the formation of a membrane composed of dense layers of connective tissue of a peculiar cartilaginous appearance upon the various serosa, viz. the peritoneum, pleura, and pericardium. The fibrous tissue forming the membrane in question may be several millimetres (0·5 to 10) thick, and forms a continuous stratified layer on the serous surfaces, having a dense opalescent appearance resembling half solidified celloidin, which may be readily peeled off the subjacent organs. . . . While no doubt chronic hyperplastic overgrowth may be confined almost entirely to the liver capsule, it is, in the vast majority of cases, part and parcel of a generalised lesion affecting by a steady progression one serous membrane after another." The process is essentially an inflammatory one, and "is almost certainly attributable to the action of micro-organisms of low virulence," although Dr. Hale White draws attention to the frequent association of granular nephritis with this condition, and alcohol in some cases appears to be a contributory factor. The condition may appear first as a perihepatitis, spreading thence to the right pleura and pericardium; at other times "the earliest signs are referable to the presence of an adherent pericardium or indurative mediastino-pericarditis, whence the process spreads to the right pleura and finally to the capsule of the liver." Thus it appears that the change affects by preference the upper part of the peritoneum, the bases of the pleural sacs, and the pericardium, and though more than one serous sac is involved they do not all present the typical opalescent or porcelain appearance which has been compared by Curschmann to the

* "Pneumonia," says Dr. Walsh ('Diseases of Lungs,' 4th edition, 1871) "is an excessively rare sequence, on the affected side especially."

† Art. "Peritonitis," by Dr. Hale White, 'Allchin's Manual of Medicine,' vol. v, p. 494.

‡ 'Reynold's System of Medicine,' vol. iv, pp. 407–9, 1877.

* "On a somewhat Rare Form of Chronic Inflammation of the Serous Membranes," 'Studies from the Royal Victoria Hospital, Montreal,' vol. i, No. 3, 1902, with bibliography.

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"icing" on a cake (Zuckerguss), various grades of the affection being met with in different regions. "The pericardium and pleura are most usually deformed by firm adhesions of the common fibrous type, and even when the hyaline membrane is typically developed it is not infrequently combined with adherent fibrous bands anchoring the affected organ to adjacent structures. Sometimes one of the serous sacs in some part of it is attacked by an acute exudative inflammation; fibrinous or fibrino-purulent exudates are not rare." In all forms of the disease the predominant feature is ascites, which sooner or later is set up and may attain enormous proportions contrasting with the general emaciation. Repeated tappings are succeeded by reaccumulations extending over some years, in this respect exhibiting a striking difference to the dropsy of atrophic cirrhosis of the liver, when the patient, according to Dr. Hale White, rarely survives for a second paracentesis. Before the ascites is established the symptoms may be referable to the chest or upper zone of the abdomen, but most cases give a history of indefinite disturbance from the first, such as anorexia, shortness of breath, and epigastric pain, or there may be actual fever with rigors, with swelling and tenderness in the hepatic region.

I have dwelt at some length on this condition partly because of its intrinsic interest and partly as being a chronic morbid state that progressively involves the serous surfaces on both sides of the diaphragm, contrasting therein with the acute inflammatory changes in these regions which, as we have seen, are more frequently confined to the site of primary occurrence.

Occasionally a right-sided pleurisy is met with in association with gall-stones, and this combination occurs "not only," as Dr. Mitchell Bruce* remarks, "as an acute complication of the presence and passage of gall-stones, that is, as acute right pleurisy following on hepatic colic, but also as a chronic process involving the base of the right chest and the right hypochondrium, when the signs may be extremely complex and difficult to interpret."

Among other features that stand out on a survey of the relations of these several inflammatory states towards one another is the very distinctly protective power exerted by the serous membranes, which are enabled within considerable limits to dispose of the infective organisms or noxious substances which may invade them, and so prevent to a great degree their extension either to the substance of the organs they invest or to adjacent serous sacs or related lymphatics. In respect to the next subject for consideration some difference from this statement will be observed.

The relation of the thoracic and abdominal cavities in respect to the incidence of tubercle is a subject of interest no less practical than theoretical. It is now fairly certainly established that the supra- and infra-diaphragmatic serous membranes may be infected the one from the other, and it is a singular fact that when these structures become the seat of tuberculous invasion, though the disease may spread from the one to the other, there is but slight disposition to extend from them to the viscera that they invest. We are all familiar with tuberculous pleurisy, its slow and insidious course, and the months or years it may be before the lungs are involved, and this limitation to the serosa is even more marked in the abdominal cavity. That there may be an eruption of tubercle on the peritoneum secondary to pulmonary tuberculosis and caseating glands in the thorax is well known, as also that the membrane may become infected from the intestinal canal by direct extension from tuberculous ulceration of the bowel, or as appears to be probable without any recognisable lesion of the intestine, the virus in such cases either passing straightway to the peritoneum or reaching the mesenteric glands, from which the serous membrane may be subsequently infected. But once given a peritoneal tuberculosis the possiblility, if not probability, of pleural invasion is to be admitted, and thus may be explained those cases of tuberculous pleurisy, which are the first evidence of intrathoracic involvement.

The mutual implication of the chest and abdomen is a circumstance that may prove helpful in diagnosis; for instance an ascites may present itself, the explanation of which may be most obscure, and attended by surroundings that may be extremely misleading in their significance, but that is cleared up when signs of pleuro-pulmonary invasion make themselves apparent. Or a patient may be ill out of all proportion to some slight basic evidences of a dry pleurisy, but this is

* "Chest Complications in Abdominal Disease," Hunterian Lecture of Hunterian Society, 1901–2.

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accounted for when the abdomen gives evidence of a tuberculous peritonitis.

Notwithstanding the direct continuity of the mediastinal connective tissue with the subperitoneal layer of the same character, an extension of inflammation and its results from one to the other is singularly infrequent. It is true that the primary affections are not common, yet mediastinitis, both acute and chronic, shows but slight disposition to pass through the openings in the diaphragm and invade the abdomen, although suppuration in the posterior mediastinum has exceptionally been known to burrow between the pillars of the diaphragm and ultimately point in the iliac or femoral region.*

A very numerous and important group of cases in which the thoracic and abdominal cavities are mutually implicated is characterised by purulent collections on one or other side of the diaphragm, constituting empyema or pyo-pericarditis in the chest, and those conditions included within the comprehensive term "sub-diaphragmatic abscess" in the abdomen.

Setting aside those rare conditions in which both cavities may be coincidently infected by pyogenic organisms, the essential interest in affections of this class lies in the diagnosis of which side of the diaphragm is the seat of the mischief or which was the primary site should both aspects be involved. The frequency with which an abscess on the abdominal side, whatever may have been its determining cause (whether a gastric or duodenal perforation, an appendicitis or an hepatic or splenic abscess), extends to the structures within the chests appears to be considerable, the diaphragm offering but little opposition to the advance of the pus. Thus out of 173 cases of subphrenic abscess collected by Lang† thoracic complications were present in 140. In 41 of these there was a serous or serofibrinous pleurisy, in 16 an empyema and in 16 a pericarditis, whilst in rather less than half the total cases there was actual perforation into the lung (34 cases), or pleura (23 cases), or pericardium (3 cases), whilst in 7 cases there were adhesions between the lung and diaphragm and ulceration through the latter. In another collection of 73 cases recorded by Elsberg perforation of the diaphragm occurred in 18, or nearly 25 per cent. A difference has been noted in the progress of those subphrenic collections of pus which are intra-peritoneal from those which are extra-peritoneal, for whilst the latter are more liable to perforate the diaphragm into the pleural cavity, the former with a slower course set up adhesions in advance and oftener rupture into the lung. It sometimes happens that the opposite side of the chest is invaded to that of the original abdominal lesion; thus an abscess of appendix origin may ultimately reach the left pleura. Although the extension of the suppurative process or the rupture of an abscess oftener takes place from the abdominal cavity into the chest, progress in the opposite direction is not entirely unknown, and pyo-pericarditis, pulmonary abscess, empyema and bronchiectasis have been found to determine a subphrenic abscess, and Meltzer has noted that the lesion may follow a croupous pneumonia without the intervention of an empyema.* It may, I think, be reasonably supposed that this difference in the direction taken by purulent collections above and below the diaphragm is mainly a matter of their following the line of least resistance, and whilst the pleural cavities or lungs are more yielding to a subphrenic abscess than the firm mass of the liver, pus within the chest may easier burst through an intercostal space or into a lung than downwards through the diaphragm. The main interest in these cases, however, is from a diagnostic aspect, and the determination of whether an abnormal area of dulness in the thoracico-abdominal region is due to fluid, and whether this be situated above or below the diaphragm or in both localities, calls forth the exhibition of great accuracy in examination and judgment in estimating the evidence obtained. Mistakes are sometimes made by even the most experienced, for no single sign can invariably be relied on. If it were possible to ascertain with certainty the position of the diaphragm the investigation would be comparatively easy, but whilst the X rays in the hands of the expert may demonstrate the movement of this organ, the rise and fall may be entirely suppressed

* Article, "Intra-thoracic Growths," by Dr. F. T. Roberts, 'Allbutt's System of Medicine,' vol. vi, p. 74.

† Quoted by Dr. T. D. Acland in article, "Subphrenic and Other Forms of Peritoneal Abscess," in Allbutt and Rolleston's 'System of Medicine,' vol. iii, 1907.

* 'Transactions of the Association of American Physicians,' vol. viii, 1893.

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when the muscular contractions are impeded by a collection of pus or other fluid above or below it, and still more when this is the case on both sides. Whilst no special difficulty exists in ascertaining the presence of a collection of fluid, pus or other, in either pleura, it is altogether different when the abscess is subphrenic. It is true that when this is situated between the right lobe of the liver and the diaphragm, the liver is most likely to be pushed down, as ascertained by the increased area of dulness and by the edge of the viscus being felt. But apart from the fact that the depression of the liver may be prevented by adhesions, the value of the evidence is discounted by the possibility of the organ itself being enlarged. Nor is the fact that an abscess in the situation mentioned raises the diaphragm and gives rise to a convex-shaped area of dulness invading the thoracic limits always diagnostic, since, as so frequently happens, the abscess either bursts into the pleura or determines a pleuritic effusion, with the result that the dull region loses its characteristic outline. So far as it goes the indication is a useful one. So also is local bulging, which, if present, is against an intra-thoracic accumulation, except in children whose chest walls are more or less yielding, and in favour of a sub-diaphragmatic abscess. It is held that when the diaphragm is pushed upwards it acts during inspiration at a greater advantage than when at the normal level, and that in consequence the range of respiratory movements are more extensive, and, as compared to the other side of the chest, are exaggerated, and that the reverse obtains when the diaphragm is depressed by fluid on its upper surface. But personally I am doubtful whether this is as obvious as it sounds. Nor is the character of the fluid withdrawn by a trocar conclusive, since a serous effusion in the pleura is not infrequently secondary to a subphrenic abscess, or the fluid may be hæmorrhagic or purulent, and this without actual perforation through the diaphragm having taken place.

Additional difficulties in the diagnosis are introduced when the subphrenic abscess contains air, as happens in a fair proportion of the cases, since the resemblance to a pyo-pneumothorax becomes very close, and both the history of the case and even the physical signs may tend to mislead, and this especially if the suppurative processes have originated above the diaphragm and perforated it. It may be that the history clearly points to an abdominal lesion and symptoms of pulmonary disease are wanting, whilst physical examination shows the lung to be normal above the region marked by amphoric or metallic breath-sounds which is not likely to be the case should it be a pyo-pneumothorax. But on the other hand there may be considerable cough and other evidence suggesting implication of the lung which may seriously complicate the diagnosis. Notwithstanding, however, it is probable that the air-containing abscess is easier of exact recognition and location than that which contains pus only, since then the signs may be exceedingly obscure, and when, as so often occurs, the subphrenic lesion has secondarily given rise to mischief within the chest the case becomes most complicated and difficult to unravel.

When the primary abdominal disease was a liver-abscess, it has been found that more than half the cases burst upwards through the diaphragm, the greater proportion into the lung, a very few only into the pericardium, and the remainder into the pleural cavity, conforming to the general rule that intra-peritoneal abscesses for the most part determine adhesions in advance of their progress, and so allow direct rupture into the lung. Now and then the hepatic abscess bursts on the surface of the organ, and this collection of pus may subsequently extend through into the pleura.

Turning now to the spread of new growths from the one cavity to the other, I have found the following results from an analysis of the tables published in the reports of the Cancer Research Laboratories of the Middlesex Hospital. During the seven years 1900 to 1906 inclusive, 790 post mortem examinations were made on patients who died in that institution from malignant diseases (carcinoma, sarcoma, endothelioma, and proliferating cystadenoma). In 477 of these the primary affection was situated in one or other of the abdominal viscera with metastatic deposits in 322, of which only 57 occurred in the thorax (mediastinal glands, lungs, pleura, heart, or pericardium). That is to say that the thorax is the seat of malignant growth secondary to that originating in the abdomen in rather less than 12 per cent. Among the many fewer cases of primary malignant intra-thoracic disease, 15 of the 790, all but one were associated with secondary deposits elsewhere, 11,

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or 73 per cent. of them, being in the abdomen. I do not wish to attach any special significance to these figures, striking as the difference is, and I give them for what they are worth, but it is at least singular that in the case of malignant growths there should be such a preponderance from the chest towards the abdomen, rather than in the reverse direction, as obtains in the other morbid states I have considered.

On this subject Dr. F. T. Roberts writes*: "Direct extension of malignant growths from the abdomen into the chest may take place, leading to secondary implication of some of the thoracic contents. Such extension is most likely to come from the peritoneum, very rarely from stomach, liver, or kidneys."

The vascular relations of the thoracic and abdominal viscera are extensive and direct, so much so that disturbance in the circulation in the one region rapidly affects the other. The relationship, moreover, is to a great extent one of adjustment, that is elaborately provided for in the course of normal existence, and that comes into play in varying degree in the course of disease. We are familiar with the relaxation of the vessels of the splanchnic area with consequent engorgement with blood that follows on peripheral obstruction in the systemic arterioles associated with spasm of the superficial vessels from exposure to cold, or with excessive contraction of the somatic muscles in the course of physical exertion, this effect being produced through the action of the depressor nerve. Owing in the first place to the enormous capacity of the liver, sufficient, it is said, to accommodate the whole of the blood of the body, and holding as it does under normal conditions about one third of the total blood quantity, and secondly to the short and direct route that exists between the right side of the heart and the hepatic vein, any obstruction that may occur to the proper passage of the blood from the right auricle, whether due to primary disease of the lungs or to difficulties at the mitral orifice or impairment in the range of inspiration, will tell back immediately on the hepatic system. The result of this is an increase in the size of the liver, which may be very considerable, and some obstruction of course to the portal circulation, which again tells back on the radicals in the whole portal area. Now the effect of all this will differ very much according as whether from the nature of the lesion the obstruction is set up very slowly and gradually or with comparative suddenness. In the latter case the liver enlargement is often not so great and is accompanied with more tenderness in the right hypochondrium, whilst in the more deliberately established condition the increase in size of the organ may become very much greater and with a minimum of discomfort. Although it may at once be admitted that portal obstruction is not by itself the cause of ascites, yet its existence is an important contributory factor in a large number of cases; at the same time marked differences are to be observed in the extent to which dropsy of cardiac origin is developed in various cases where at first sight the conditions in regard to the obstruction appear to be very similar. I have myself been disposed to attribute this difference to the variation in degree of engorgement of the liver, the incidence of the dropsy being deferred, or of slighter amount when this reservoir-like function of the liver is more slowly and more extensively brought about, since it would seem that a large quantity of blood might thus be locked up as it were in the gland without producing so much block in the area of portal drainage.

From a clinical point of view much the most interesting relationships between the thorax and abdomen are those in which, with structural disease in one region, there is a purely symptomatic involvement of the other without any gross anatomical change. It will be readily perceived that the existence of such conditions may, and as a matter of fact not infrequently does, lead to considerable difficulty and mistakes in diagnosis.

This simulation of abdominal by thoracic disease is no matter of recent discovery, but has long been known; its recognition, however, has only assumed much importance within the past few years, since indeed laparotomy has become a frequently performed operation for the treatment of many abdominal affections that otherwise are all but invariably fatal.

A large number of cases have now been placed on record—and indeed they are of sufficiently frequent occurrence to have come within the purview of most of those with hospital experience—of acute thoracic disease that have presented during

* Article "Intra-thoracic Growths," 'Allbutt's System of Medicine,' vol. vi, p. 93, 1899.

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the first twenty-four hours, or even more, marked symptoms referable to the belly, whilst those more properly pertaining to the actual malady are overshadowed by these abdominal indications or have not yet developed. As might perhaps be expected, it is morbid states of the lower zone of the thorax and adjacent to the diaphragm that exhibit the features now referred to, and it may be at once said that it is only a small proportion of such cases. What it is that determines this distal reference of symptoms in some cases and not in others still remains to be explained. Basic pneumonia, pleuro-pneumonia, empyema, pyo-pneumothorax are some of the conditions that have been observed to simulate most closely certain abdominal diseases, more particularly gastric irritation of a severity that suggests irritant poisoning, appendicitis, acute peritonitis, perforation of a gastric or duodenal ulcer, or it may be even the passage of a gall-stone or some other acute malady of liver or biliary passages. Notwithstanding that the chest affection has been known to develop subsequent to severe injury to the thoracic walls, even to causing fracture of the ribs, the immediate resulting symptoms have been almost wholly abdominal, and have even led to a diagnosis of peritonitis for which laparotomy has been performed. So close may the resemblance be that not only are pain and tenderness, often of an acute character, experienced over the abdomen generally, or more distinctly limited to certain situations such as the epigastrium or cæcal region, but there is associated rigidity of the abdominal muscles and marked diminution of the abdominal movements of respiration, whilst the belly becomes distended from paralytic dilatation of the intestines. There may be nausea, vomiting, hiccough, and constipation, and the similarity to peritonitis and other grave abdominal maladies may be further increased by the manifestation of collapse with cold sweats and pallor, and even of the characteristic appearances comprised in the term *facies hippocratica*. Under such circumstances it is not to be wondered at that the surgeon's aid is invoked unnecessarily, and it becomes therefore of paramount importance to ascertain if there be any indications that may prevent so regrettable an incident. Fortunately careful observation and the due estimate of certain phenomena should serve, at least in most cases, to furnish what is required. To begin with, it is seldom that definite local signs within the chest are delayed more than twenty-four hours in making their appearance after the more or less sudden onset, although, of course, it is within this time that operation when actually required has often the best chance of success. Probably the most dependable evidence in favour of a given case being essentially thoracic rather than abdominal in nature—though co-existence of disease in both cavities is not out of the question—is the pulse-respiration ratio, the rate of the breathing being often as high as 40 per minute with a pulse not exceeding 100 and perhaps much less. Such a condition should at once give pause, and calls for the most careful examination over the bases of lungs. Sometimes, too, the peculiar short and "catching" character of the inspiration may be noticed, which is specially suggestive of pleuritic involvement. Certain features of the rigidity of the abdominal muscles and of the tenderness have been pointed out as differing from what obtains in acute peritonitis, for in these pseudo-peritonitic states the muscles may be felt to relax between the respirations, which are almost entirely affected by the costal movements, and whilst superficial contact is provocative of extreme tenderness, firm and deep pressure may be well tolerated, especially if the patient's attention be diverted. This is in striking contrast to the hyperalgesia of acute peritonitis which some degree of pressure is usually required to elicit, and where the skin alone may be sometimes pinched up without discomfort. Most of the cases included within the group now under consideration exhibit a higher range of temperature than that usually met with in the early hours of acute peritonitis, 103° or 104° F. being not infrequent. The diagnostic significance of local swelling in the cæcal region detected, it may be, by rectal examination, should not of course be overlooked.

The explanation of this manifestation of symptoms remote from the actual site of production is to be found in the nervous connections. Intrathoracic lesions such as pleurisy involving filaments of the lower dorsal nerves may give rise to symptoms referred to the areas of distribution of other branches of those nerves; or sensory impressions originating in the lungs or heart are carried by the splanchnic afferent fibres to the sympathetic ganglia and thence by the white rami to the posterior root ganglia of the lower dorsal nerves, where they come

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into relation with the somatic fibres distributed (as previously described) to the integuments and muscles of the abdominal wall as well as to the parietal peritoneum. Thus is accounted for the muscular rigidity (spasm from irritation) with pain and tenderness that may be experienced over the region of the appendix, suggesting disease of this structure when branches of the tenth and eleventh dorsal nerves are implicated in a pleurisy, or it may be that the symptoms are mainly referable to the umbilical district which corresponds to the area of the tenth dorsal nerve, or higher still to the epigastrium and so resembling a gastric ulcer or perforation, or more towards the right hypochondrium with a likeness to mischief in the liver or gall-bladder, both of which are regions served by the seventh, eighth and ninth dorsal nerves. The connection of these several nerves with the sympathetic system by means of the rami communicantes indicate the way in which the various associated symptoms arise, such as nausea, vomiting, diarrhœa or constipation, vascular phenomena and other evidences of collapse.

That individual symptoms referable to the abdominal viscera are likely to be set up in the course of definite structural disease within the chest is not to be wondered at when the nervous connections are remembered. As illustrating this may be mentioned the vomiting so apt to follow on excessive coughing as from whooping-cough or pulmonary phthisis, and reference may be made to the severe epigastric pain and sickness that sometimes immediately precedes death in cases of aortic valvular disease.

Similarly many symptoms of disturbed pulmonary or cardiac action arise, which are really in origin to be referred to some affection of organs below the diaphragm, and yet sufficient, perhaps, in their intensity to completely mask their source and give good reason to suppose that it is heart or lung which is at fault. It is, however, particular symptoms or combinations of them that are so referred; nothing in the nature of a simulation of a definite thoracic malady by any condition within the abdomen takes place comparable to the imitation of appendicitis by pneumonia, or a perforated gastric ulcer by acute pleurisy.

Foremost among such are disturbances of cardiac action, whether in rate, force, or rhythm, accompanied, it may be, by sensory manifestations of an anginous character. Of all these palpitation is probably the most frequent, but an unduly depressed action of the heart, amounting in extreme cases to syncope, may be the accompaniment of certain abdominal disorders, whether of so temporary a character as a gastric indigestion, or of so serious a nature as the passage of a calculus, the perforation of a hollow viscus, or a general acute peritonitis. In these cases the direct influence of pain, which has a marked effect in modifying cardiac and also respiratory action, has to be taken into consideration. Attacks of true angina pectoris, occasionally and very much more frequently of that form of cardio-respiratory discomfort known as pseudo-angina, are attributable to over-distension of stomach or bowels by gases, or even by an unduly large meal, and enlargement of the liver has been in rare cases looked upon as the determining cause. It should be observed, however, that cardiac and respiratory disturbances, associated as they commonly are, should be regarded very often as concomitants of flatulence rather than as consequences, the gastro-intestinal defect as well as the dyspnœa or abnormal heart's action being alike due to some common cause.

The respiratory disturbances of abdominal origin are equally well marked, although, perhaps, scarcely so well known, since they are on the whole less frequent. As examples one might mention the so-called "stomach cough" as an expression of a gastric or gastro-intestinal dyspepsia, but a similar manifestation is far from unknown in connection with uterine affections. A true dyspeptic dyspnœa is sometimes observed, together with other symptoms of indigestion, or so predominating as to obscure its real origin. Along with a sense of oppression and impeded breathing is a constant desire to take a deep inspiration, occurring in attacks often of some hours' duration and not infrequently at night—a condition which recalls in its description a fit of asthma, as a provocative of which indigestion also takes a high place. Paroxysms of this strange complaint are often directly attributable to some dietetic error in quantity or quality, or to time of meal in respect to sleep, or are associated with flatulent distension of stomach or colon, whilst more rarely the presence of intestinal worms has been held responsible. It is noticeable, however, that it is with functional derangements of the gastro-intestinal tract rather

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than with structural disease that asthmatic attacks are likely to be associated.

Without doubt the explanation of these referred symptoms, as of those occurring in association with the abdominal organs in primary thoracic disease, previously considered, is to be sought in the nervous connections, at least for their immediate causation. At the same time, besides reflex excitation there is another factor that in all probability plays some part, and perhaps an important one, though it also most likely produces its effect *viâ* the nervous system. We are well aware that cardiac or respiratory disturbances may be set up by the introduction into the body of various poisons, and there is good reason to believe that toxic bodies with similar properties may be produced in the course of indigestion, whether gastric or intestinal. The brilliant work of Starling and Bayliss, which has shown that the excitant to glandular secretion is in many cases of a chemical rather than of a nervous character, and brought about by substances termed by them "hormones," has not, so far as I am aware, extended to showing that muscular action is capable of being induced by similar agents, with the possible exception of adrenalin; nevertheless, the stimulus to muscular activity may not only be of a purely nervous character, since the direct application to the contractile substances of reagents without the interposition of nerve-fibres or terminals may suffice to produce response. It may be that certain toxic products of indigestion or of perverted metabolism in liver, pancreas, kidney, ovary, or uterus may act likewise.

I have thus, gentlemen, within the limits of my time, discussed some of the relations—causal, pathological, and symptomatic—that obtain between diseases of the chest and abdomen. In doing so I am conscious that I have advanced nothing fresh, and, indeed, have only recounted what must be familiar to all of you. Nevertheless, I hope I have not wholly wasted your time in directing your attention especially to the morbid states of that particular region which lies on the confines of the territory with which it may be presumed in an institution of this character you are principally concerned. Of the importance of the subject there can be no doubt, especially from the point of view of diagnosis and, consequently, of treatment, and whilst in the pursuance of the former all your technical skill will be called for in the eliciting of data by physical examination, you will equally have to interpret phenomena that are less capable of such precise investigation. As contributory to this I have endeavoured to set before you, maybe in somewhat too artificial and didactic manner, the main facts concerning those diseases in which chest and abdomen may be mutually involved.

*June 29th*, 1908.

# A CLINICAL LECTURE
## ON
# RHEUMATOID ARTHRITIS.

Delivered at the Great Northern Central Hospital.

By H. E. SYMES-THOMPSON, M.A., M.D.Cantab., M.R.C.P.Lond.,
Physician to the Hospital; Assistant Physician to the Royal Hospital for Diseases of the Chest.

THE study of chronic joint diseases has been handicapped by the complexity of the nomenclature. "Osteo-arthritis," "rheumatoid arthritis," "arthritis deformans," "rheumatic gout," and "chronic rheumatic arthritis," are some of the terms with which the literature abounds, and these and other names have been used by different authors when describing similar conditions. It is advisable to drop most of them, and with the American authors to retain provisionally "osteo-arthritis" and "rheumatoid arthritis."

Osteo-arthritis will signify that process of later life in which certain joints become enlarged, painful and creaky. It is the disease of eburnation, lipped cartilages, and osteophytes. The term "rheumatoid arthritis" will be confined to a less common condition only superficially like the above. The following is a short clinical description of it. Rheumatoid arthritis typically begins as a more or less acute joint affection in young women accompanied by constitutional disturbance. The joints first attacked are usually those of the hands, and extension tends to take place to other joints and to cause permanent deformity and crippling There are thus two stages in the disease, the early or active stage, and the chronic stage or stage of deformity. In the active stage the joints of the wrists, hands, and fingers are swollen and hot, the palms are sweating and cold, the skin may become thin and shiny, and the nails may degenerate The joints of the feet and of other parts may rapidly become affected. A striking wasting of the muscles in the neighbourhood of the diseased joints is noticeable, and patchy pigmentation of the skin may be found in different parts of the body. The axillary and inguinal glands are sometimes enlarged. The pulse is quickened, and there is marked anæmia and depression of spirits. In favourable cases almost complete recovery may

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now take place. But as a rule the subsidence of the acute symptoms marks the beginning of the chronic stage in which the joints tend to become more and more fixed.

There is some evidence that rheumatoid arthritis has a nervous origin, but the theory that it is due to poisons circulating in the blood which attacks joints, the resistance of which is lowered, has more support. This accounts both for the constitutional and the local disturbances. There is often a history of loss of blood, leucorrhœa, insomnia, or prolonged lactation, and worry or some specific fever precedes the onset of the arthritis in a considerable number of the cases. Moreover, micro-organisms have been found in the joints by some observers, though it should be stated that they made no differentiation between cases of rheumatoid arthritis and osteo-arthritis. Max Schüller* found bacilli constantly in the inflamed synovial membrane, and in the hypertrophied fringes. He sometimes found cocci also, but regarded them as secondary, like the cocci which are not uncommonly found in the joints of gonorrhœal and other infective arthritides. The bacilli were uniform in their characters, and when they were injected into the joints of rabbits a condition followed similar to that in the human joints from which the bacilli had been isolated. Ballantyne, Wohlmann, and Blaxall,† almost always found a short bacillus in the joints of these cases and occasionally also in the blood. Poynton and Paine ‡ found diplococci and *Bacillus coli communis* in the joints of an arthritic patient. They cultivated the diplococci, injected the cultures into a rabbit, and two days later arthritis appeared in several joints. When the animal was killed a white flaky fluid from which diplococci were cultivated was found in the affected joints. The heart was normal.

The improvement in the joints which follows the treatment of pyorrhœa alveolaris or vaginal discharge is often remarkable, but in the majority of cases no primary focus of infection can be discovered. When this is so our attention should be directed to the alimentary canal. In this connection the discovery of *Bacillus coli communis* in a diseased joint by Poynton and Paine is suggestive. And as we shall see when treatment is considered, the results which we can claim from drugs are referable to those which inhibit the multiplication of micro-organisms in the intestine.

A post-mortem examination on a young woman suffering from rheumatoid arthritis in the acute stage, who died from an inter-current malady has been published by Hale White. There was fibrous thickening outside the joints, and the synovial membranes were swollen and pink. Fluid was present in excess. No erosions such as those found in osteo-arthritis were found, and there were no osteophytic outgrowths. In the bone itself the changes were slight, being obviously secondary to the joint inflammation. Numerous post-mortem examinations have been reported on cases of rheumatoid arthritis in the chronic stage, and a careful study of the condition of the bones and joints has been made by Strangeways.* The atrophied muscles are found to be contracted, and owing to this and to the proliferation of the soft parts the joints are more or less fixed. The final step is that the cartilages and bones atrophy from pressure, and fibrous or bony ankylosis may be the result. No eburnation or osteophytic outgrowth is found even in long-standing cases.

The diagnosis is made from osteo-arthritis from its special incidence upon young women, and from its onset as a subacute joint inflammation with anæmia and constitutional disturbance. Skiagrams show it to be a disease of the soft parts with a tendency to atrophy of the bones as is shown by rarefaction, while in osteo-arthritis we have lipping and osteophytes and destruction of the joint structures. Chronic gout is commoner in men, there is a history of acute attacks with tophi and deposits in the joints, and sodium biurate is present in the blood. Skiagrams show sclerosis of bone instead of rarefaction, and certain spicules known as Bruce's nodes. Chronic rheumatism is also commoner in men. There may be a history of acute rheumatism or some allied condition, and it has no preferential joints. Gonorrhœal arthritis tends to affect many joints, and to settle in one of the larger ones, and there is a primary focus such as a urethritis in which the gonococcus may be found. It should also be remembered that secon-

* 'Berl. klin. Wochenschr.,' 1896.

† 'Lancet,' 1896, vol. i.

‡ 'Path. Soc. Trans.,' 1902.

* 'Bulletin of the Committee for the Study of Special Diseases,' Cambridge, 1907.

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dary syphilis is sometimes accompanied by a general arthritis with constitutional disturbance.

[The lecturer here showed several patients and skiagrams illustrating many of the points referred to.]

A favourable prognosis may be given if whole-hearted treatment can be instituted at the outset. If, on the other hand, the case does not come under observation until the chronic stage is reached, the disease will usually prove to be progressive

Rheumatoid arthritis is a disease of weakness, and the treatment must be of a tonic and supporting nature; the patients should be placed in good hygienic surroundings, and be shielded from worry. Any infective focus such as a vaginal discharge should be treated. In the acute stage rest to the affected joints must be insisted upon. The hands are to be lightly splinted, and the patient confined to a couch. Massage for the wasting muscles is beneficial. In the chronic stage we have to deal with joints more or less permanently damaged and cure is impossible. But much may be done by means of regular massage and movement to mitigate the crippling of the joints, and by climatic and other treatment further extension of the disease may be prevented. There is a liability to relapse, and the joint movements should be remitted when such a suspicion suggests itself. Moderate exercise is to be encouraged, even with a persistently rapid pulse. The diet should be liberal throughout. The drug which has been followed by most benefit is guaiacol carbonate. It should be given in full doses for six months, and the improvement which follows may be explained on the assumption that it inhibits the growth of certain micro-organisms in the digestive tract, with a consequent diminution of the infection of the blood-stream from the intestine. Iron and arsenic may be used to combat the anæmia. When the pain is severe aspirin often gives relief, and methyl salicylate may be painted on the joints. An old remedy for the pain is to surround the joints with bags containing hot sand, and remarkable results have been reported recently from Bier's method of passive congestion. This consists in tying a ligature round a limb sufficiently tightly to hinder the venous but not the arterial flow. Some œdema is produced, and the application is made for from six to twenty-two hours. One application often relieves the pain for several weeks, but unfortunately the effect usually diminishes with repetition. Patients with rheumatoid arthritis are very susceptible to cold, and it is found they do best in warm, equable climates, such as Egypt and the Canary Islands. Baths should be ordered with caution as they tend to be depressing, though brine baths may be spoken of with less reserve. Mud baths are useful in relieving pain and stiffness, and there is nothing to be said against the trial of vapour baths locally applied to the joints.

*June 29th*, 1908.

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**Varicose Veins of the Papilla of the Kidney a Cause for Persistent Hæmaturia.**—W. F. Whitney reports the case of a middle-aged man with a history of repeated attacks of hæmaturia lasting over a considerable period of time. Examination by modern methods as well as in the usual clinical way revealed no cause for the trouble until finally bloody urine was observed coming from one ureter. The corresponding kidney was removed. It appeared pale but otherwise normal. The tips of one or two of the papillæ were a little reddened, and under the microscope these tips showed the small veins, dilated irregularly to twice or three times their normal size, and covered on their free surface with the finest possible film of connective tissue and a single layer of epithelial cells. Here and there they projected as varicose knuckles above the surface. The conditions, in fact, were such that the slightest violence must have caused a rupture with subsequent escape of blood. In one specimen a rent was found in one of these thin-walled channels, but it cannot be stated positively that it may not have been an artefact. The lumen of the vessels in many places was filled with a finely granular detritus and occasional blood-corpuscles, but no well-marked thrombi were seen. That hæmorrhage must have taken place here, however, is borne out by the fact that the straight tubules of this region contained blood-corpuscles, which were not found higher than the base of the pyramid.—*Med. Rec.*, vol. lxxiii, No. 23.

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## A CLINICAL LECTURE

ON

# ACUTE TUBERCULOUS PERITONITIS IN ADULTS.

Delivered at St. Bartholomew's Hospital.

By W. P. HERRINGHAM, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—On February 27th this year a young woman, æt. 22 years, was admitted into Mary Ward, looking very ill. She was a hospital nurse, employed in private nursing in Yorkshire, and had come to London on February 16th or 17th to a nursing institution in order to train for maternity work. The day after she came down, that is to say on the 18th, she felt giddy, though on the next day she felt quite well. It was not until a day after that, namely, February 20th, that she was taken with fever and headache and a bad sore throat, and general malaise. She was in very good hands, being in a training-place for nurses. She was immediately put to bed, and we have an account of her symptoms from the day of the onset of the illness. She got gradually worse, and remained in a condition of very high fever, varying between 100° and 105° F. for a week. Various things were tried, but nothing seemed to do her any good. In addition to the symptoms I have mentioned she had diarrhœa. At last, on February 27th, they brought her to us at this hospital. When she was admitted we found she had in the first place a sore throat, secondly, high fever (it was 103·4° F. when she was admitted), and besides, a pulse varying from 96 to 112 in the day, slight friction below the right nipple, abdominal distension, and some tenderness chiefly on the right side of the abdomen. The diarrhœa persisted. I should have said that the abdominal pain and distension had been noticed

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on the third day of her illness, before she came into this hospital. When you have a patient with fever which has been lasting for a week or more, with distended abdomen and diarrhœa, the first thing you think of is what we first thought of, namely, typhoid fever. But it was not a typical case of that disease. The history was not right for that. Typhoid fever does not begin with this sudden onset and high fever from the start. Such a temperature as 105° F. on the first two days of typhoid fever is practically unheard of. If this was typhoid fever the only thing we could suppose was, that although she was a sick nurse, and although she had been trained to observe other people, she had not been observing herself —she had missed her own history. So it was with considerable doubt we entertained the idea of typhoid fever. Widal's reaction was found to be negative. Besides, there were no spots, and there was no enlargement of the spleen. There was nothing, in fact, to point to typhoid fever except the fact that she had had fever for a week with diarrhœa. If it was not typhoid fever, the next thing we thought of was, could it be one of those diseases which are called " para-typhoid " diseases, in which, though the typhoid bacillus is not the cause of the illness, yet a very similar set of conditions is produced by something, that something being generally a bacillus resembling the *Bacillus coli?* We tried her serum first of all against typhoid bacilli, and several strains of it, and then we tried it against para-typhoid disease, but in neither case was there any specific reaction. The next thing that we asked ourselves was whether, with a sudden onset of pain in the abdomen, it might be pneumonia. That occurred to me, but it might not have occurred to you, because you do not realise until you have made mistakes two or three times what a great degree of abdominal pain patients with pneumonia may complain of. It is common for people to locate their pain in the left side of the abdomen when they have really inflammation of the left lobe of the lung. I remember a case in which a girl was brought in with no history except that of violent sudden pain in the abdomen. She was exceedingly ill and rather collapsed, and she was actually operated upon for supposed perforated gastric ulcer. She had nothing but pneumonia. I knew that case perfectly well, and yet it was only the other day that I made the same mistake. I was called to see a case in Kenton Ward for Mr. Rawling, in which a man was complaining of severe pain and tenderness in the abdomen. I knew he had pneumonia because I made that out, but the pain in the front of his abdomen was so great that I thought it could not be due merely to the pneumonia, and that there must be peritonitis as well. I warned Mr. Rawling that I thought it might be pneumococcal peritonitis as well. But the man had nothing but the ordinary pneumonia. Sometimes such symptoms are due to the presence of pneumococcal peritonitis as well as ordinary pneumonia. Only the other day Dr. Drysdale had a little boy in Mary Ward in whom there was both ; the child had pneumonia of the lung and also suppurative pneumococcal peritonitis. He died after some weeks. Was our case a case of that sort? It was not. In the first place, our patient had no signs of pneumonia. In the second place, she had not the aspect of that disease, that is to say, that form of peritonitis which occurs with pneumococcal disease, or with septic disease. She had not pneumonia, neither did I think she had pneumococcal peritonitis or septic peritonitis. If you have seen cases of the ordinary septic peritonitis which follows, for instance, appendicitis or perforation or obstruction of the bowel, you will know that besides the common symptoms of peritonitis, namely, pain and tenderness to pressure and distension of the abdomen and rigidity of the abdominal walls there is also extreme prostration and collapse ; the patient looks very ill. This girl did not look a bit like that. The pain was not very great, neither was the tenderness, and she certainly was not in the least collapsed by it. Then another symptom came in. In an ordinary case of septic or pneumococcal peritonitis unless the patient is moribund the blood invariably contains a large number of leucocytes : unless the patient is wholly unable to resist the disease and is going to die very shortly, his blood reacts at once and he produces a large leucocytosis. There was not that in this case. Her leucocytes were only 4600, which was even below the normal level. So that, taking all things together, we came to the conclusion that she could not have any ordinary peritonitis. But the tenderness, the distension and the hard feeling of the abdomen, made me feel sure that she had some form of local inflammation of the peritoneum.

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Now, it so happened that fourteen years ago I had seen a case that impressed me very much. A young woman, æt. 26 years, healthy looking and well nourished, came into our hospital on April 10th, 1894, with the following story: That on the 24th March she had felt slight pain and some shivering; that next day she had come to town and met her husband, who was a sailor, on his return from sea. The pain had got worse, but it was never sudden or severe. There had been no vomiting, and she had not even been obliged to go to bed until she came into the hospital, although she had not been able to get about and enjoy herself as they had meant to do. She had been treated with aperients. There was no cause that she knew for the attack, and in going into her family history we found nothing in reference to tuberculosis in any member of the family. When she came in on April 10th there was tenderness all over the abdomen, but no ascites; there was limited movement, and the abdominal wall was to some extent rigid. But the pain was not at all acute; she was not crushed by it as in septic cases. That is the phrase I find in my notes written at the time. No local cause could be found, and *per vaginam* everything was quite correct. There was no evidence of disease in any of the pelvic organs. She became worse, and on April 20th, ten days after she was admitted, the abdomen was opened. Blood-stained fluid was found in the peritoneum, and the omentum presented itself in the wound as a great thickened mass. It was recognised that the condition was either tubercular or malignant, but nobody could say which at the time. Nothing was done and the wound was stitched up. Two days later she died. The peritoneum was found to be studded with miliary tubercles, the omentum matted and thickened, the intestines adherent, there was a small ulceration in the transverse colon, evidently secondary to the original disease, and every other organ in the body perfectly natural. The original focus from which the tubercle was disseminated could not be found, though it was very carefully looked for. The temperature up to the date of the operation had varied between 101° and 104° F., very irregular, hectic. After that it was normal. I remembered that case, and I always have remembered it, because I had never seen anything like it before, and I had not seen one since until this case came in. And on the first day that she was admitted it struck me as being like the case in 1894, and I said so. And of course, with the presence of tuberculosis, the low leucocyte count was quite consistent. There was no other evidence to make one suppose that it was tubercular disease, except that we had found a little friction below the right nipple. Now, in adults, friction without pneumonia—in other words, a little local pleurisy—is very suggestive indeed of tubercular disease. Pleurisy, with or without effusion, unless there is some very marked cause for it, is always suggestive in adults, and especially in young adults, of the beginning of tubercular disease. And eventually, a few days later, she began to develop a little more evidence in her lungs: we found râles, first at one apex and then at the left base, and those became irregularly scattered over the lungs, and some of them are there to this day. And after much search in her sputum, which was a very laborious search, one tubercle bacillus was found. Now, in 1894, the date on which I saw the first case I mentioned, we had no other means of diagnosing tubercle but the presence of the tubercle bacillus. But nowadays there is the further test of the reaction of the patient to the injection of tuberculin. That can be done in two different ways, as you all probably know. In the first place it can be done by injecting what is called old tuberculin under the skin, and in the second place by the Calmette reaction—that is to say a drop of Calmette's tuberculin solution is placed into the conjunctiva of one eye. Her hectic temperature forbade us trying the old tuberculin injection. We could not have seen if any reaction had taken place. But we did try the Calmette reaction, and it was slightly positive. So those things taken together clinched the diagnosis of tuberculous disease, and therefore of tuberculous peritonitis. And that was subsequently proved to be correct by the condition that developed in the abdomen. In place of a slight rigidity and a suspicion of ascites, of which we never had felt quite sure, there has gradually developed a great solid mass running in the direction of the attachment of the mesentery, from the tip of the tenth left costal cartilage right away towards the cæcal region. Those of you who are my clerks have felt that great thickened ridge stretching in a slanting direction across the abdomen. That is very typical indeed of the

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tuberculous adhesion that forms in tuberculous peritonitis. It is an adhesive form; the intestines become completely matted together, and it is they, I think, which form this great transverse ridge which is so characteristic of tuberculous disease in the peritoneum. These cases of tuberculous disease of the peritoneum in adults are not at all common. If you look at Osler's book on medicine you will find it stated that the greater number of cases of tuberculous peritonitis occur between 30 and 40 years of age. I cannot conceive that that can be correct, unless it includes all cases that post-mortem are found to show tuberculosis of the peritoneum. But for practical and clinical purposes, what we want to know is how often does a case occur in practice in which the disease as it presents itself to us is tuberculous peritonitis? Because a case coming with phthisis which subsequently gets a little pain in the abdomen is, of course, of no clinical importance. And if you take our statistics of cases returned as tuberculous peritonitis, that is to say, cases in which the tuberculous peritonitis was the substantive disease, then out of 130 cases which have occurred in the last ten years there have been only 25 which were over twenty years of age. All the others were under twenty, and that exactly coincides with my own experience, which is that tuberculous peritonitis is very rare except in children or very young adults. The cases are not always quite like this one. We had in my ward the other day a different form of the disease. Two years ago a young fellow, æt. 20 years, was admitted to Colston Ward with extreme rigidity of the abdomen and local pain. His story was that for a month he had been having sharp pain after his meals, and that he had been losing flesh and getting worse. In his case there was a family history of phthisis, On his admission there were signs of tuberculosis at one apex of his lung. The abdomen was rigid. and there were present from the first lumps in the left side. Those lumps became diffused all over his abdomen, so that he had an abdomen which felt almost solid. He became gradually worse and eventually died. In that case the disease was from the first extremely painful, and it was characterised by much more tumour formation than was the case with the girl I have just described to you. I believe that those are the only two cases of tuberculous peritonitis as a substantive disease that I have ever seen before in adults over twenty years of age. So I can assure you it is an uncommon thing.

Now we come to the treatment, which in this case was very interesting. In a great many cases, especially those which are characterised by ascites rather than by fibrosis, it is very good treatment indeed to open the abdomen. They do exceedingly well after that. Whether it necessarily is because of it I do not think is quite proved. But I have seen several cases in which it looked as if the operation had actually led to the cure, simply opening the abdomen and draining off the fluid. In one case I remember the operation could hardly have cured the actual peritoneal disease, because the peritoneum was not opened. A little girl was admitted under my care for ascites, and she had also a lump in the abdomen. An operation was being performed on the right side of the abdomen to open the peritoneal cavity when it was found that the lump which was thought to be inside the abdomen was actually in the abdominal wall. The surgeon who was performing the operation felt unable to remove the tumour in the abdominal wall, as it was very diffuse and went into the tissues a considerable distance. He believed it to be a sarcoma. He therefore simply closed up the incision which he had made, without having entered the peritoneum. That child got perfectly well of her disease—proving of course that it was not malignant. She died three years later and we saw her on the post-mortem table. Her death was due to tubercular meningitis, and we found old obsolete tubercles, which had dried up, in the peritoneum. In that case, therefore, it was not the opening of the peritoneum which cured the disease. But in very many cases laparotomy appears to do a great deal of good. But not in those cases in which the disease is characterised by fibrosis and large tumours. You cannot do any good by it; there is no fluid to let out and you cannot remove the tumours, and you are very likely, in opening the abdomen, to simply find a mass of adhesions, to which you can do nothing whatever. Those cases do not improve after the operation. In this case I did not entertain any question of operation, and there was never any definite sign of fluid although at one time we suspected it. At first we tried treatment simply by drugs, in which we were greatly hampered by the fact that the patient was con

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tinually sick. We tried quinine, hydrocyanic acid, and bismuth, but each drug we gave we had to stop because of sickness. We tried creasote and cod-liver oil, but had to leave that off, and then gave quinine and iron again, and then guaiacol carbonate. At last I began to treat her with injections of serum. She was first given injections of horse serum—that is simply non-specific serum drawn from the horse—10 c.c. in each dose, and a very minute dose of the new tuberculin, namely, $\frac{1}{20000}$ of a milligramme injected under the skin. I show you the temperature chart. At the time we began the treatment it was not followed by any great result. We began to give the treatment on March 28th. After the second injection of horse serum on April 1st she had a brawny induration at the site of the injection. It hurt her very much. This injection is sometimes very painful in adults when it is given. She also had a very high, hectic temperature. We were unable to try anything of the sort for a few days, when she again had horse serum, this time by the mouth, as has been recommended by Dr. Latham, of St. George's. I went on with the tuberculin hypodermically, as that is a very minute injection and does not hurt. She had horse serum on April 7th, and tuberculin on April 9th. Each was repeated at intervals of about a week. Notice the gradual fall of temperature, which has been maintained up to now: up to March 28th it was often 103° F.; from April 9th to 20th it was only on five days over 101° F.; from April 21st to May 4th it was only four times over 100° F.; for the last week it has been normal. This treatment is purely empirical; it is on its trial, but it gives one hope of being successful so far as the temperature goes. But I think we must wait before we can say that anything like a cure is going to result in this case from the treatment. One would like to see as the result of it something which has not yet happened, namely, an increase in the weight of the patient and the signs in the lungs completely clearing up. So far as this one symptom, that of the temperature, goes, the improvement is quite obvious; but we need to have other indications of improvement as well.

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# GENERAL SUPPURATIVE PERITONITIS.

A Post-Graduate Lecture delivered at the London School of Clinical Medicine.

By LAWRIE McGAVIN, F.R.C.S.Eng.,
Surgeon to the Dreadnought Hospital, and Lecturer on Clinical Surgery in the London School of Clinical Medicine.

GENTLEMEN,—The subject I am discussing with you to-day has, I may say, little that is new about it; but as few conditions exist in the whole range of surgery or medicine which can present to you as general practitioners greater difficulties of diagnosis in the earlier, or of treatment in the later stages, or which can cause you more anxiety in your practice than that of general suppurative peritonitis, I have chosen it as the subject of this lecture.

It must have struck you that there are few things more remarkable in the surgical progress of the last twenty-five years than the change which has come over both our ideas and our methods with regard to the surgery of the peritoneum. Formerly a region of the direst menace to patients, and of the greatest apprehension to surgeons, it is now a region in which every day, operations are undertaken with the fullest confidence in the reparative power of this same membrane; and from being a bogey of the most pronounced type it has become a very tame servant of the modern surgeon. This statement is, however, only partially true, and although by deliberate intention the greatest liberties may be taken with the peritoneum, accidents to the patient's internal organs may convert it into the scene of a conflagration, which in many cases the surgeon is powerless to arrest.

The occurrence of general peritonitis may be said to depend on two primary conditions, viz. injury plus bacterial invasion, and bacterial invasion alone. Needless to say the former is by far the commoner, the latter only occurring as the result of specific micro-organisms carried from certain parts of the body other than the intestinal tract. Thus it occurs secondarily to pneumonia and to gonorrhœa, and has been seen in the course of such conditions as acute nephritis and acute articular rheumatism; the invasion of the peritoneum during acute infectious diseases without the

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traumatic element of ulceration is, however, very rare, and the condition known as "idiopathic" peritonitis probably does not exist as such.

The list of possible causes of infection of the peritoneum is naturally a long one, and I do not propose to go into these causes with you in detail; but I should like to give you some idea of the relative frequency with which they are responsible for the trouble. Benda, investigating this question in 446 cases post mortem, obtained the following results:

| Originating in the— | |
|---|---|
| Appendix | 115 cases |
| Stomach } Duodenum } | 68 ,, |
| Intestine | 118 ,, |
| Female genitals | 81 ,, |
| Gall-bladder | 10 ,, |
| Kidney } Bladder } | 10 ,, |
| Pancreas | 2 ,, |
| Spleen | 1 case |
| Post-operative | 4 cases |
| Hæmatogenous | 2 ,, |
| Unknown | 35 ,, |
| Total | 446 |

And I think that this list fairly represents the facts as investigated by other observers.

Appendicular abscess, gangrenous appendix, and perforated gastric ulcer are certainly the commonest causes of peritonitis. Among the cases arising in the rest of the intestinal tract, the majority are accounted for by typhoid ulceration; while among the unknown causes, one is at liberty to suppose the existence of an "idiopathic" peritonitis, but the cause is much more likely to be found in some slight lesion of the intestinal canal which has been overlooked. Quite recently I had occasion to operate upon a patient who, three days previously, had complained of acute epigastric pain. This was soon followed by vomiting, tenderness, and rapid pulse, and when I was called to her on the third day of her illness she had every sign of general peritonitis. There was a history of previous gastric ulcer, and as the pain was referred to the epigastrium I opened the abdomen above the umbilicus. To my surprise I found nothing the matter with the stomach; it appeared to be in every respect normal. On returning it and pressing back the intestines, pus began to well up from below. I then opened the abdomen over the appendix, released more pus, and found the appendix normal. I then explored the pelvis and found nothing whatever to help me. The pus was sweet and was therefore probably not from an intestinal lesion. In spite of washing out and free drainage the patient died. No post-mortem examination was permitted, so I am unable to tell you the cause of the trouble; this case, however, would probably have been put down years ago as "idiopathic" peritonitis. I have no doubt that had an examination been made some demonstrable lesion would have been found.

Let me tell you of another case bearing on this point—a case of very practical importance. A few weeks ago a seaman was admitted to this hospital suffering from acute abdominal symptoms which suggested, rather indefinitely, perforation of the appendix. He had been ill only about twelve hours and his pulse had not as yet risen beyond 80, but his abdomen was very rigid, he was in great pain, and was vomiting at intervals, his general appearance being typically abdominal. I opened his abdomen over the appendix and found a large quantity of slightly turbid fluid with flakes of lymph in it; it was unlimited by adhesions, and there was nothing to point to its point of origin, the appendix being to all appearances perfectly normal, as was also the lower end of the ileum. As the greatest quantity of fluid seemed to me to be about the appendix I suspected rather than diagnosed it as the cause of the trouble, and removed it. On opening it I was surprised to find it extensively ulcerated. There had been no perforation, and therefore I can only suppose that the trouble arose by transudation, and that the infection was toxic rather than bacterial. The abdomen was washed out and drained, and the patient quickly recovered.

Before proceeding further, I would like to remind you of certain points of interest in connection with the peritoneum which may help to explain the well-known gravity of general suppurative peritonitis.

Nature presents in certain parts of the human body problems of construction which at first sight appear enigmatical: of these the knee-joint is one, and the peritoneum another. In the former we have the curious fact of the most dangerously

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complicated joint in the body being guarded by a capsule, part of which is made of bone, the structure of all others in the body least capable of bearing sudden strains; and further, this bone is placed in one of the most prominent positions for the reception of injury, and has to withstand the action of the most powerful lever in the body.

In the latter, the peritoneum, we see the most delicate membrane, the most remarkably absorptive surface, and the most acutely sensitive structure closely applied in every direction to a tube whose contents are of the most virulently poisonous character, and from which, in the event of perforation, it has no means of escape. Again, into this membrane in women, opens a mucous tube capable of conveying to the abdominal cavity one of the most virulent of the micro-organisms which can attack it.

The area of the peritoneum is very remarkable, and can be reckoned in square yards, and taken with its wonderful power of absorption easily explains the rapid onset of the symptoms of general peritonitis. In its arrangement I would have you look upon it as the lining of a house having an upper and a lower story, the floor between them being represented by the transverse meso-colon and great omentum, and of these two the peritoneum lining the former is most sensitive and absorptive, the close proximity of the semi-lunar ganglia, the aortic plexuses, and the lymphatics of the phrenic region making it a very dangerous situation for suppurative processes. Again, the multiplicity of its shelves, partitions, and pockets makes it very difficult to wash out and drain; and lastly, the rapidity with which it will throw out a protective covering of lymph, although in slight cases of infection a very great safeguard and localising agent, may, in the case of general suppuration, become a positive danger, since it shuts up infective material in various corners, and holds bacteria in its meshes, rendering the process of peritoneal cleansing almost futile.

With regard to absorption, Muscatello's experiments showed that the peritoneum is capable of taking up in one hour over 3–8 per cent. of the body weight, but that under toxic influences it can throw a similar proportion of exudate into its cavity, while Dubar and Remy were able to recover granules of carmine from the thoracic duct seven minutes after intra-peritoneal injection.

Muscatello further pointed out that lymph spaces exist only beneath the diaphragmatic peritoneum, and that these spaces are capable of transmitting solid particles; and he proved the presence of a normal lymph current in the peritoneal cavity, always towards the diaphragm, independently of the position of the patient.

The virulence of general peritonitis depends on various factors, and of these the chief are—the amount of the initial dose, the situation first affected, the portion of the intestine originating the trouble, and the character of the infecting material. Thus gastric and gall-bladder perforations, owing to the nature and amount of their discharge, and the fact that they are situated in the upper part of the abdomen, generally result in a peritonitis of a very fatal type. Lingood and Cushing have shown that the upper portion of the intestine is much poorer in bacterial flora than that lower down, and that partial starvation is capable of rendering the former comparatively sterile; therefore a small perforation in the upper portion in starved patients is a much less serious matter than one of equal dimensions in the large intestine of one who has been for some time constipated.

The particular variety of bacterium in greatest preponderance has also much to do with determining the virulence of the infection, even although commonly many different organisms are present. Of these bacteria, probably the most virulent are the *streptococci* and the *Bacillus coli*, while the least so is the *Staphylococcus albus*; this organism is, however, often a concomitant of the first mentioned, as are also the *S. aureus* and *citreus*. In some cases it is very difficult to say what the organism causing the original trouble was, but in perforation due to typhoid ulceration Klein and Korte failed to find typhoid bacillus in the pus, although the latter found it in the peritoneal pus resulting from the suppuration of a mesenteric gland. Therefore the causative organisms were ousted presumably by others of greater vitality inhabiting the intestine.

By many investigators the *Bacillus coli* is looked upon as a bacterium of marked primary virulence, and Orlowski, who isolated eleven different varieties, found that injection of living cultures or their toxines into the healthy peritoneal cavities of dogs and guinea-pigs resulted in invariable peritonitis.

The gonococcus and the pneumococcus are of

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themselves capable of generating a general suppurative peritonitis, the former giving rise to a particularly virulent form of the disease. In a few cases the *B. ærogenes capsulatus* has been the organism at work; and in one case Flexner has reported the presence of *B. proteus vulgaris*—a bacillus commonly believed to be non-pathogenic to man.

The varieties of peritonitis originated by these organisms have been conveniently arranged by Pawlosky into four classes, as follows:

(1) The rapidly fatal type which he has called "mycotica," and in which death occurs before any inflammatory reaction is established.

(2) The hæmorrhagic type, in which a serofibrinous exudate accompanies the formation of peritoneal ecchymoses. This is the variety commonly seen in perforative cases.

(3) The sero-purulent type, in which a thin serous exudate is formed containing much lymph deposit.

(4) The true purulent type, in which the exudate consists of much pus of the consistency of thin pea-soup, together with flakes of lymph.

But of course all these types run more or less in combination. Age is an important factor; peritonitis arising from similar sources in an adult as in a child is, owing to the much more intensely absorptive character of the membrane in the young, of far greater virulence in the latter. The amount of the infecting material and the degree of initial shock will largely depend on the state of the viscus from which the trouble arises, while the extent of the distribution of such material is largely influenced by the position of the patient at the time of infection. Thus the perforation of a large gastric ulcer while the patient is in the erect position, and after a full meal, will probably produce a rapidly fatal result; whereas, that of a smaller lesion occurring during sleep in the recumbent position, and while the stomach is empty, may lead to the localisation of the pus, or at worst to an unlocalised infection of a much milder type than the last. Again, it is a notable fact that, other things being equal, general peritonitis in a robust individual is often a more rapidly fatal condition than when it occurs in one who has been some time ill. The explanation probably depends on the extremely active absorption of which the healthy peritoneum is capable, as against that of one in which the splanchnic circulation is enfeebled; as well as upon an opsonic index of low degree compared with one which has possibly been raised by the nature of the disease preceding the catastrophe.

In considering the signs of general suppurative peritonitis, it must be at once admitted that although the cardinal symptoms are well known, they are often unaccountably absent. Indeed, in few conditions are they so liable to be mis leading. Let us consider them briefly.

Pain, being a purely subjective sign, and therefore liable to exaggeration in neurotic subjects, must be very carefully investigated. First, its site of origin should be noted, as this will often give a clue to the cause when there is no reliable history to help you. Then its character is important; any tendency to intermittence, to spread from its original site, or to increase from the first should be considered. Intermittence is suggestive of colic rather than of inflammation; spreading of the painful area and increase are indications of the latter. The onset is variable; in perforative cases it is commonly sudden and agonising, while in others it is slow and therefore less valuable as a sign.

Tenderness, which always accompanies general peritonitis, is of greater value as a sign. It should accompany the pain and be especially marked in the area of its origin, and as it at times assumes the character of cutaneous hyperæsthesia, may help you the more easily to note any tendency to spreading. If it is absent in the presence of pain, there is good reason to suspect the absence of peritonitis.

Rigidity of the abdominal walls you should, I think, look upon as a very important sign, since it is perhaps the least easily simulated for any length of time by neurotic subjects, in whom it is not uncommonly seen. It is of most import when it commences in the originally painful area and gradually involves the whole abdominal wall. But it should be continuous from the first, up to the time of onset of meteorism.

Vomiting, when it is repeated from time to time, is rather a sign of peritoneal irritation than of inflammation, and of this the following case is a good example:

Some years ago I operated upon a patient in the Hospital for Women, for gall-stones. The gall-bladder was very contracted and I could not bring it nearly up to the abdominal wall; I therefore drained it across the peritoneum by a tube,

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and packed some strips of gauze beneath it in order to catch any leakage. For the first three days the patient was persistently sick, and her pulse was over 125 for 48 hours, her temperature being normal. It all looked very like peritonitis, but her abdomen was not rigid nor was there any tenderness except in the immediate vicinity of the wound. I concluded that the gauze was at the bottom of the trouble and removed it, after which she immediately ceased to vomit and made a perfect recovery.

Vomiting is, however, seldom or never absent in purulent peritonitis, and when accompanied by eructations and persistent hiccough may be looked upon as a cardinal sign.

The position of flexion of the hips and knees is, I think, of less importance than some writers would have us suppose. It is the posture assumed by most of us in bed, but if on extension from this position the patient complains of an increase of abdominal pain, then it is of value.

Facies abdominalis, or facies Hippocratica, is more marked in suppurative peritonitis than in any other abdominal lesion, and the explanation of the pinched features, the sunken eyes, and the cyanotic lips is to be found in the abstraction of fluid from the subcutaneous tissues, and the deficient aëration of the blood from failing circulation, and respiration hampered by abdominal pain.

At times the pulse and temperature give rise to doubt, the former being under 100 and the latter raised; such a combination, however, is of commoner occurrence in localised peritonitis, or in some case where the infection, although unlocalised, is of a mild type. The temperature is of far less importance than the pulse; and where the latter shows a tendency to gradual acceleration the indication is a very grave one. Of all the signs of abdominal catastrophe, none are so significant as that of a rising pulse accompanying a falling temperature, and in few cases is this truer than in that of general suppuration of the peritoneum.

Inability to pass flatus and motion, the retention of copious evacuant enemata, and the appearance of meteorism are very important signs, and suggest either paresis or obstruction. If, however, peristalsis can be seen actively in progress, there can be no question of peritonitis; the case will probably prove to be one of obstruction.

I have left two signs to the last, viz. the appearance of meteorism, and the diminution of pain. They are the later signs, and to my mind of very grave import, both of them signifying that the case has passed into a deeply toxic condition from which recovery is unlikely. As regards the former it will only appear in the early stage where it is due to actual free gas in the abdomen due to perforation. The statement of the patient that she or he is no longer in pain and is much more comfortable, in the presence of other signs mentioned, is an indication almost of fatal prognosis; it is at times accompanied by a dusky mottling of the abdominal skin.

Of the conditions which most closely resemble peritonitis, there is one which from its close mimicry must always head the list, for fear it should be overlooked and lead to an unnecessary operation. It is that of abdominal neurosis in women, and to show you the close resemblance I will read you the notes of a case recently admitted to this hospital.

The patient was a woman, æt. 39 years, and was admitted with the history of acute abdominal pain coming on forty-eight hours previously, and gradually spreading from the right iliac fossa over the whole of the abdomen.

On admission her temperature was normal and her pulse 100. There was marked tenderness in the right iliac fossa, and to a less extent over the whole of the lower portion of the abdomen. This part showed persistent rigidity and was motionless, while the upper part, although rigid, was moving slightly on respiration. Her tongue was dry and brown, the lips were covered with sordes, and the whole aspect of the patient was abdominal. She was, moreover, frequently sick, and had retained a turpentine enema, and had passed nothing in the way of wind or motion for two days.

On her abdomen being opened over the appendix nothing whatever was found to account for her signs, either here, in the pelvis, or elsewhere.

Again, intestinal colic may be confounded with peritonitis as it is accompanied by agonising pain, rigidity of the abdomen, and at times by vomiting; the pain is, however, not continuous, comes on in spasms which repeatedly cause an increase in the pulse-rate, and is usually unaccompanied by tenderness, in fact, pressure commonly relieves it.

Renal and biliary colic owing to the vomiting

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and abdominal pain may cause mistakes, but even when the pain and tenderness cross the middle line of the abdomen, the attacks are paroxysmal, the pulse drops between attacks, and there is an absence of intestinal paresis. A little patience and justifiable delay will differentiate the conditions.

Intestinal obstruction, especially when not due to such chronic conditions as might suggest its occurrence, may closely simulate peritonitis. But there is the cramp-like spasm, the obvious peristalsis, and usually the localisation of the pain and tenderness to help one; and the fæcal character of the vomit, which, when it does occur in peritonitis, is only seen in the last stages as the result of extreme meteorism.

Acute gastro-enteritis and uræmia may be mistaken for peritonitis, but you should have little difficulty in distinguishing them: the diarrhœa in the first, and the suppression, fits, and slow pulse of the second should point the diagnosis.

Acute hæmorrhagic pancreatitis may very closely resemble peritonitis, since it is ushered in by vomiting, pain, distension, cessation of peristalsis, with increasing collapse, rapid pulse, and low temperature. An error of diagnosis in this condition is very commonly made. There is, however, a tendency for the tenderness to be at first localised to the epigastrium, and thus at this stage there is a suggestion of perforated gastric ulcer. The picture is certainly that of an abdominal catastrophe, and will in any case point to the necessity for laparotomy.

Lastly, it must not be forgotten that certain pulmonary conditions, as pointed out by Barnard, at times closely mimic peritonitis. They are such conditions as diaphragmatic pleurisy, basal pleuro-pneumonia, and pyo-pneumothorax of the bases. He points out that the symptoms are due to the gastro-phrenic distribution of the phrenic nerve, and that close observation will help you to note that they are thoracic as well as abdominal; the respirations being in excess of what is customary in peritonitis, and being accompanied by a slight but distinct catch at the top of each inspiration. Once suspected, the stethoscope will help you to clinch the diagnosis.

If in any case you should still be in doubt, do not forget the value of leucocytosis. This phenomenon has been more studied abroad than in this country, and although of the greatest value in many cases, surgical opinion of it as a general evidence of pus formation has been somewhat modified. Thus intestinal obstruction, especially if cancerous, usually shows a moderate leucocytosis, and therefore this method cannot be depended upon alone for a differential diagnosis. It is, moreover, of little use in mild cases of catarrhal appendicitis, in salpingitis, or in acute gangrenous appendicitis; and very slight increase of leucocytosis is present in uncomplicated typhoid fever, malaria, and tuberculosis. On the other hand, a general or localised suppuration produced by the ordinary pyogenic cocci usually results in a markedly increased leucocytosis. Dunham has drawn attention to the fact that all aseptic surgical procedures are followed by a considerable increase, especially where ether has been used as the anæsthetic, and he quotes eight cases in which the highest, shortly after operation, was 29,000. He further points out that in certain individuals there is a digestive leucocytosis of 10,000 to 13,000 a few hours afrer a full meal. In young children there is often a physiological leucocytosis of 10,000 to 12,000. The condition is seen also after large hæmorrhages. This observer is of opinion that in progressive suppurations the qualitative changes are of importance, a larger or smaller proportion of polymorphonuclear neutrophiles staining with iodine according to the activity of the process.

To the practical surgeon, then, leucocytosis may be of the greatest value in certain circumstances; and these I would state as follows: In young adult life, middle, and healthy old age, where there is no reason to suspect constitutional diatheses, a persistently high or gradually increasing leucocytosis affords, in the presence of abdominal symptoms, strong presumptive, although not conclusive evidence of suppuration.

There is no doubt that systematic estimation of leucocytosis can be of very great assistance; and the fact that in a few cases it has proved fallacious is no good reason why it should not be carried out as a routine practice.

Now, gentlemen, when referring to works of reference as to the treatment of general peritonitis, you must, I am sure, have been struck by the great variation in mortality statistics given by various writers. The discrepancy probably arises from the fact of a misconception of the term

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"general." It is obvious that if we are only to consider those cases as localised in which the septic area is definitely bounded by adhesions, and are to look upon everything else as general peritonitis, our statistics will vary enormously. For it is certain that many suppurations, although unconfined by adhesions, may be operated upon in the pelvis before the poison has spread to the supra-colic region, or in one loin before the pelvis or other loin has been affected. Such conditions are not truly general, and therefore the prognosis is more favourable. The least that I would be prepared to accept as justifying the term "general" would be the involvement of the pelvis, loins, and small intestine, *i.e.* the infra-colic region of the abdomen. The mortality of a peritonitis of such extent is very high indeed, probably little short of 85 per cent. even when well treated. Where the disease has involved the supra-colic region as well, the mortality may well rise to 90 per cent. to 95 per cent., presuming that the ordinary pyogenic cocci are the cause.

Seeing, then, the dangerous condition with which you have to deal, it is perfectly clear that the earlier your treatment is commenced the better it will be for your patient: and you may look upon the first forty-eight hours as the time of grace in which your efforts are most likely to be crowned with success. Laparotomy is, of course, the only reasonable treatment; but cases occur in which there may be a doubt as to which quarter of the abdomen is to be investigated first. Remember the difference in absorptive energy and sensitiveness of the upper and lower compartments of the abdomen. Supposing you have opened the abdomen below the umbilicus, and found traces of pus but nothing to account for it either in the pelvis, loins, or among the intestines, you may be tempted to explore the upper region of the abdomen, only to find no sign of peritonitis there; you have thus put your patient to the risk of an extension of the trouble to this part, your hands having already been in the infected lower compartment of the abdomen. Where there is a doubt then, explore the upper sensitive region first: you will add little to the shock, and you will thus avoid a danger which is otherwise inevitable, no matter how careful you may be.

Next explore the most probable seat of the trouble—the appendical region; not through trap-door incisions, they are out of place here, since, drainage being necessary, primary union is impossible, and therefore whatever effect the trap-door incision might have had in the prevention of a hernia is obviously destroyed. The appendix may be dealt with if found diseased, but if not the hand is passed into the pelvis, and the pelvic organs are examined. Finally, the lower end of the ileum may be sought and brought out of the wound if there is any reason to suspect a perforation; the character and odour of the pus will, however, help you to a supicion of this from the first.

Having, then, found and dealt with the cause of the trouble, or possibly failed to find it, you will proceed to cleanse the abdomen. Two methods are still in use for this purpose; the first, chiefly employed in this country, viz. very thorough washing out of the cavity with saline fluid, without any exposure of the bowels; the second, still much advocated in America, that of complete eventration followed by extensive lavage, sponging with gauze, and picking off of any loose flakes of lymph.

That the latter is a decadent method in this country is, I think, admitted; it is, I believe, a very serious cause of collapse, for the mesentery is pulled upon in the cleansing, the bowels are at times almost impossible to return owing to the distension, the removal of lymph is tedious and at times causes oozing of blood, and the difficulty of keeping the great mass of intestine at the proper temperature is very great. It is by no means certain that the layer of lymph adhering to the intestines is not in some sense a protection from absorption of septic material, so possibly harm is done by its removal.

Lavage from within the abdomen, then, is the safest method; the saline used should be at a temperature of 110° to 115° F. *on reaching the abdomen*, and should flow in freely. Now, if the upper part of the abdomen is unaffected, you must be careful to prevent, as far as you can, the fluid from being forced up into it; accordingly you must ensure a perfectly free return for the washings before you commence; and this is best done by using large drainage-tubes passed through the left iliac and supra-pubic regions, while with your hand in the right iliac wound you keep the bowels in motion, and prevent them from stopping up the tubes. With this hand you can place the irrigation

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tube in any position you desire, and thus cleanse every corner of the abdomen. If possible, the patient should be placed in the semi-recumbent position, so that the washings run down into the pelvis.

When the upper portion of the abdomen is also involved, it is very necessary to cleanse the subdiaphragmatic space: the tube is therefore passed through the epigastric wound and carried back between the liver and the diaphragm, along both sides of the falciform ligament, and along the gastro-hepatic and gastro-splenic omenta. The fluid thus rushes down through the abdomen to the pelvis, and leaves it by the three wounds already mentioned. Much good may be done by leaving the irrigation tube in position here, as it will act as a drainage-tube, and at the same time the abdomen may be flushed out once or twice in the first twenty-four hours from it. This will not only help to cleanse the cavity, but will stimulate the patient, dilute the poison, and keep the drainage-tubes from becoming clogged.

As regards drainage, other methods may be used than the employment of tubes of glass or rubber. Thus, simple gauze strips may be employed, but have the disadvantages that, once in, they adhere quickly to the intestine and so are difficult to remove; once removed it is difficult to replace them with fresh gauze; and lastly, their tendency to cause the formation of adhesions is well known. A better method is a combination of the tube and gauze; wide glass tubes of Keith's pattern are passed into the wounds, one into the pelvis, and one to either side of the mesentery, and down them are passed wicks of cotton or selvedge gauze loosely packed, which wicks may be changed several times in the twenty-four hours. At the end of the third day, if all goes well, the two iliac and the epigastric tubes may be removed, and the supra-pubic tube on the following day.

It is interesting that Clark, of Johns Hopkins University, noting the presence of the subdiaphragmatic lymph spaces, the ready absorption here, and the normal lymph current towards this part, concluded that the correct attitude after a laparotomy in which there had been any risk of a subsequent peritonitis, was that in which the pelvis was raised, so that any exudate might flow to this region. It has since been shown, however, that although these spaces will take charge of moderate amounts of septic material, they soon become clogged by leucocytes, bacteria and *débris*; and all recent observations go to show that it is this very region which we must at all costs shield from infection.

The position recommended by nearly all surgeons to-day is that in which any accumulation will tend to gravitate towards the pelvis. And here I may tell you of a practical point with regard to the treatment of acute appendicitis threatening early perforation: anticipate as far as possible the rush of septic material which may come from the part, by placing your patient in such a position that if rupture takes place the pus will gravitate to the safest region: this position will be upon his right side, so that the infection may be as far as possible limited to the right iliac fossa. In the case of threatening gastric perforation, the less upright the patient is kept the better, since the lower portion of the abdomen would in this case tend also to become affected; therefore probably the supine position is the best here.

The very great drain which septic peritonitis imposes upon the fluids of the body must be met by one of the methods of saline infusion. Of these there are four: the intra-venous, the intra-cellular, the intra-peritoneal, and the rectal. Personally I prefer the former to the others, first, because it is more rapid in its action, and secondly, because the intra-peritoneal is unsatisfactory where the abdomen has been drained. But as its effect is rather evanescent, it should be supplemented by one of the other methods after operation, and the best is certainly that by continuous infusion into the rectum, the fluid being fed to the rectum at the rate of about half a pint per hour, till three or four pints have been absorbed. The addition of adrenalin chloride to the intra-venous saline is a very valuable one, and a drachm in each pint will be found to alter the tension of a running pulse in a very remarkable manner. Adrenalin chloride is, however, not so suitable for rectal use, since it is inclined to blanch the mucous membrane, and so prevent the rapid absorption of the fluid.

The use of sera in the treatment of general peritonitis has as yet hardly become a recognised method, probably because the infection is so frequently a mixed one; but where the organism chiefly concerned is the streptococcus, the anti-streptococcic serum should be used and vigorously pushed, being given in doses of 20 cc. to 40 cc.

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twice daily. When the *Bacillus coli* is the organism present in greatest numbers, a difficulty arises from the fact that there are many different strains of this bacillus. Dudgeon and Sargent have, however, recently produced a serum containing a multivalent antitoxin, with which they seem to have had some success in the treatment of general peritonitis of intestinal origin. The method is, of course, still *sub judice*, but the following points may be of use to you.

The dosage of the serum, which was produced in the Wellcome laboratories, ranged from 20 cc. to 40 cc., and no ill-effects have been noted after as much as 40 cc. in a single dose. In all cases it appears to have had a beneficial effect on pulse and temperature, has induced sleep, and in some cases appears to have had a localising effect on the peritoneum. The cases reported by Dudgeon and Sargent numbered twenty-five; all were of the worst type, two of them having recovered after having had "black vomit"—usually a sign of fatal prognostication—for over twenty-four hours. Of the whole number, nine recovered and sixteen died.

For the relief of the pain in early peritonitis little can be done except by the use of morphia, and this it is undesirable to give, unless the diagnosis is certain and operation has been determined upon, for fear of obscuring symptoms. The application of hot fomentations of large size may, however, give some relief; and here I might express the opinion that we are not nearly active enough with such treatment in most cases. The benefit derived is mainly due to the heat of the fomentation, and to some extent proportionate to the area over which it is applied. It is, therefore, unreasonable to expect much good to come of a fomentation of small size, thin consistence, and infrequent application. On the abdomen it should be large enough to cover the whole area, should be composed of four layers of lint, and should be changed at least once every hour. One which is half the size of the abdomen, two layers thick, and which is changed according to the hide-bound rule of every four hours, is much more likely to thoroughly chill, and so result in the death of the patient, than in the relief of his pain.

The question of purgatives is still a debated one, and although the old practice advocated by Lawson Tait of giving a strong saline purge at the commencement of a general peritonitis is still, I think, an excellent one, it must be given *at the beginning* of the trouble only; once the inflammation has become established the bowels must be kept perfectly quiet; and in the late stages the paresis of the intestine cannot be overcome by drugs, so that the presence of purgatives will only give rise to discomfort and do no good.

That stimulants must be freely administered goes without saying, and strychnine and digitalis hypodermically and brandy by the rectum may help considerably. The vomiting is very difficult to control, and the mouth becomes parched and dry and the lips covered by sordes. I have found nothing better for these discomforts than iced champagne containing two or three minims of liq. strych. hyd.; it is at least as useful in some cases as cocaine, iodine, ingluvin, cerium oxalate, and other remedies. At the same time it must be admitted that the control of peritoneal vomiting is a matter of the greatest difficulty, and more often than not, if unrelieved by operation, persists up to the time of death.

*July 6th*, 1908.

**Apex Tuberculosis in X-ray Pictures.**—Normally the apex presents a black shadow with whitish streaks made by the vessels. The pathological findings may be as follows: There may be well-marked diffuse shadow without the blood-vessel streaks; the shadow may be evident only in the upper portion of the apex and present a linear demarcation from the rest of it; cloud-like, often confluent spots may be seen; distinct, separate, lens-like foci; distinct deep shadows, probably due to calcified spots; unilateral white border running parallel to the lower edge of the second rib. The sources of error are found in pictures showing no contrast; in over-exposed plates, which show the vertebra very distinctly, while the apices appear as very dark shadows without vessel streaks; in under-exposed plates, where bone and lung tissue cannot be differentiated; in lack of sharpness in the pictures. Other sources of error are found in the local changes of the tissues overlying the lungs, and in the bad position of the patient during the process of taking the pictures. Only technically perfect X-ray pictures are useful in the diagnosis.—*Med. Record*, vol. lxxiii, No. 24.

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# WITH MR. STUART-LOW AT THE CENTRAL LONDON NOSE, THROAT, AND EAR HOSPITAL.

## Alar Collapse and Narrow Nares.

It is exceedingly tantalising, not to say exasperating, to the rhinologist to encounter on the very threshold of his special domain such an intractable, unamenable, and hitherto, even in many instances, incurable condition as that of alar collapse and narrow nares.

This serious nasal defect often co-exists with other disturbances of the normal nasal interior; and the specialist, having overcome these, finds his best efforts thwarted, and all his skill and labour lost, because the nasal impediment may still persist in the form of collapsed alar cartilages or narrow nares. This seemingly simple impairment will often frustrate every other endeavour to effectually remove it, and compel resort to the insertion, and necessity of wearing, some annoying and irritating mechanical appliances to assist respiration through the narrow anterior nasal entrance.

It is often and forcibly impressed on anyone having much experience in nasal operative work that, should collapsed alæ nasi or narrowed anterior nares, either one or both, exist, with substantial obstruction in the nasal interior, such as deviation of the septum, for instance, and should, as is usual, the obstruction inside the nose alone be operated upon, relief will not be obtained so long as the entrance is left untouched. The writer has, on the other hand, had patients on whom he has simply operated, after the method here described, on the narrowed nares, and left the interior obstruction alone; and the respiratory relief has been so great that the patient has refused further and necessary operative measures for the septal displacement, even when it has been of very pronounced obstructive character.

It is no uncommon experience, therefore, to meet with instances where adenoids have been removed, or the posterior ends of the inferior turbinals snared off, and yet the expected improvement in nasal respiration has not followed, because the anterior nasal aperture has remained permanently narrowed from the alar collapse persisting. It will not be so surprising, then, to learn that *even* where the most thorough, efficient, and skilful removal of septal displacements by Killian's method has been done good results have not been obtained, and much disappointment, both to operator and patient, has been experienced, through the first and most obvious cause of nasal obstruction being overlooked, viz. alar collapse or narrowed anterior nares.

This is a matter intimately concerning the medical practitioner, as he frequently recommends patients to have operations performed for embarrassed nasal breathing, and much of the opprobrium, if the operation is a failure, must naturally recoil upon his undeserving head.

It behoves the rhinologist, therefore, to consider this question, and to endeavour to discover if some way cannot be found over the difficulty, and something done to relieve or remedy such a distressing factor, preventing, as it may, the realisation of perfect freedom of nasal breathing.

The normal anatomy of the lateral nasal cartilages is very remarkable, and affords an eminent example of a perfect mechanism for the maintenance of a patent and resilient anterior nasal aperture.

The chief framework consists of four lateral cartilages—two upper and two lower. The upper plates are flattened and triangular in shape, with their apices above and the bases below. The inner borders of these plates are in apposition, and supported by the cartilaginous septum mesially. To the bases are attached the upper convex margins of each of the lower lateral cartilages. The lower lateral cartilages are laterally elongated plates of thin cartilage, with a convex outer surface, and each meets its fellow in the middle line, thus mainly forming the apex of the nose.

Each of these lower lateral cartilages has two limbs—an outer, constituting the cartilaginous ala nasi, and an inner, participating in the formation of the central columna nasi. This inner wing only extends half or two thirds of the length of the columna nasi, and ends in a rounded, pointed portion, which often projects quite visibly from the outer side of the central nasal column. The cartilaginous framework of the nasal entrance is completed by two more groups of structures, viz. the sesamoid, or segmental, cartilages, and the mesial end of the nasal septum.

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The termination of the cartilaginous nasal septum in front varies much in the amount that it contributes to the formation of the columna nasi. There is an important and very special manner in which this septal cartilage may become a cause of nasal narrowing, viz. when its broad and flange-like, fluted, anterior border is well developed. All its borders—the posterior, the lower, and especially the anterior—have a winged formation, and when this is so in a considerable degree on the anterior and inferior borders, a distinctly symmetrical thickening can be seen projecting from the side of the columna nasi, and can better be felt if the columna nasi is grasped between the finger and the thumb.

The sesamoid cartilages are small plates clustered around the outer edges of the entrance to the nose inferiorly. They help to complete the space between the outer end of the lower lateral cartilage and the base of the columna nasi.

In many cases of alar collapse and narrow nares the septal terminal cartilage pushes its way between the two lower wings of the lower lateral cartilages and forces them apart. This has the effect of narrowing still more the entrance to the nose by giving rise to bulging outwards of its inner margins. I have operated upon bad cases of alar collapse in which there existed not only such separation of these inner wings by the central cartilage, but where there was also a flange-like broadening of the sides of the separating cartilage.

Firm fibrous tissue also enters into the formation of the boundaries of the nasal apertures; it binds those cartilages together, and, by filling up all gaps, rounds off and completes the entrances.

In most of such cases that I have examined and operated upon, these two natural anatomical features have been much exaggerated, viz. the broad rim of the anterior end of the cartilaginous nasal septum, and the spur-like projection of the end of the inner wing of the lateral cartilage on the columna nasi. Together these two enlargements cause the columna nasi to be broader than normal and give rise to a narrowing of the anterior nasal openings. There is also in alar collapse not only a falling in of the outer limbs of the lower lateral cartilages, but a projection and twisting outwards of the inner limbs of this cartilage. These two alterations readily give rise to a narrowing of the anterior orifices of the nose, and constitute the real obstruction to free respiration.

Broadly speaking there are six variations in the character of the narrowing brought about by these departures from the normal.

There may be merely the alar collapse—the falling in of the outer limb of the lower lateral cartilage (first degree); or the chief condition constituting nasal narrowing may consist of alar collapse *plus* marked projection of the pointed end of the inner limb of the lateral cartilage (second degree); or alar collapse *plus* broadening of the septal cartilage in a flange-like way (third degree); or alar collapse *plus* 2 and 3 (fourth degree); or alar collapse *plus* prolongation of the septal cartilage between the inner lateral wings of the lower cartilages (fifth degree); or a combination of all of these (sixth degree).

The method that I have been practising for some time, and found best for overcoming the obstructions caused by these enlargements, and which I advocate in this paper, consists simply in removing them by subcutaneous and submucous resection.

As before remarked, I have seen but few cases where the alar collapse presented itself purely and simply, without at any rate the termination of the inner lateral cartilage turning out into the already narrow passage. When such is the case I still operate on what there is to be found of the inner limb, since my experience is that removal of this portion, and firm stitching up tends to throw out the outer limb and so widens the entrance to the nose.

A crescent-shaped incision is made, commencing above on the side of the columna nasi, just anterior to the projection made by the inner limb of the lower lateral cartilage, and passing alongside of this projection, which can be easily defined with the manipulating fingers of the disengaged hand. The incision ends at the lower angle of the nasal aperture and is entirely through the skin.

A second incision beginning almost exactly where the first did is carried downwards behind the protuberance on the columna nasi nearly to the same position as the first; it may skirt the protuberance as in the first incision, so as to excise a piece of skin of the width, at its widest part, of $\frac{1}{6}$th of an inch. It may be necessary to remove half as much skin again as this if the protuberance is

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very large and the skin is redundant. This marked out piece of skin with its subcutaneous tissue is then dissected away. In some cases it facilitates the operation, when the skin is very adherent to the underlying cartilage, to remove the exposed cartilage at the same time. This is freely done, it being trimmed down to a very thin piece.

The soft structures are now reflected from the lower end of the nasal septum, and this cartilage is also freely removed. The broadened border is always excised, and the septal cartilage for about $\frac{1}{4}$th of an inch beyond it.

The chief reason for taking away more than the mere projecting part of the septum is, that this tends to let the upper nasal lateral cartilage sink down, and in such a manner leads to slight flattening of the nose. This pushes the outer limb of the lower lateral cartilage out, and so broadens and widens the nasal entrance.

The incision is then firmly stitched up with catgut. The healing takes place by first intention. This is always so if an oiled-silk dressing with firm plugging is applied during the first twenty-four hours (see 'Lancet,' May 4th, 1907).

Considerable manipulative dexterity is required to perform this operation neatly and nicely. I have done most of my cases under local anæsthesia, and no general anæsthesia seems necessary; but this depends upon the person, and it might be better to administer a general anæsthetic in the case of some very sensitive people. In a usual way, however, the patient simply sits in an upright position, and has the head sufficiently supported. The exterior and vestibule of the nose are first sterilised with a solution of ethereal soap, or a weak solution of lysoform, and then sponged with a 1 in 2000 solution of spirit and biniodide of mercury. The parts are rendered anæsthetic by applying repeatedly a 10 per cent. solution of cocaine by means of cotton-wool mops, and a pad of wool soaked in this solution is applied for ten minutes. This usually completes the anæsthesia, but if it is thought desirable to make it more perfect, a submucous injection of eucaine or cocaine may be made.

If the enlargement of cartilage on the columna nasi is symmetrical, as is almost always the case, then it is necessary to repeat the excision on the opposite side; but in the majority of cases one can easily reflect off the mucous membrane from the septum, and complete this part of the operation through the primary incision.

On the removal of the plugs the feeling of freedom of breathing is immediately experienced, the patients frequently giving generous expression to their sentiments on the pleasant change.

This is emphasised on exertion, as a greater current of air can be with facility inhaled.

A young man sent to me for nasal obstruction from the Chest Hospital exemplified this very well. He was in advanced pulmonary phthisis and was considered such an unfavourable case two years ago as to be refused admission to a sanatorium, being unlikely to gain permanent benefit.

He had well-marked alar collapse and also a considerably deviated nasal septum. The septum was not operated upon; but this operation for narrow anterior nares was performed, with the result that his breathing was very greatly improved. At home, his rooms being up four flight of stairs, he can now ascend without having to rest two or three times, as was formerly the case.

If properly performed, there is not the slightest visible mark after this method of operating, and no inconvenience, such as perforation of the nasal septum, which results in some other procedures. Not only are the impediments to free breathing removed from the inner side of the entrance, but the outer wing of the orifice becomes everted so that the unfavourable indrawing on deep breathing is lessened.

The victims of slit-like anterior nasal orifices have in the large proportion of cases the nose in other respects well developed. The bridge is usually high and the nose prominent and thin, being pushed up to a high degree by the septum.

The sesamoid cartilages frequently lie too close to the base of the septum, and if pushed outwards and slightly upwards can be seen to cause the alæ to protrude. The palliative means often employed to overcome these defects consist of putting a ball of wool into the upper recess of the vestibule, and instructing the patient to practise expansion of the alæ by forcing into action the dilator-nares muscles. I have found that fitting on a truss at bedtime and wearing it during the night gives valuable assistance in restoring the proper position of the cartilages. One limb of the truss fits under the sesamoid cartilage on each side, and the other takes the form of two pads placed over the upper lateral cartilages.

The effect of this is to somewhat broaden the narrow nose and expand the alæ.

I have said that a certain skill and nimbleness of manipulation is essential to success in performing this operation, and I would, lastly, add a word of caution to any whose enthusiasm might exceed their experience, calling their attention to the great risk of making a perforation if scrupulous care is not observed, since the structures are often exceedingly thin and delicate.

*July 6th*, 1908.

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## WITH DR. JAMES TAYLOR IN THE OUT-PATIENT DEPARTMENT OF THE NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC.

THIS man complains that his sight began to fail about eight months ago, and that it has become worse lately, and now he is unable to see at all. He tells us he had always been quite healthy before then. He is married, and declares he has never had syphilis. His pupils react definitely to light, *i.e.* there is no Argyll-Robertson pupil. But on examining his discs one finds that he has very distinct optic atrophy. He has no lightning pains. On testing for his knee-jerks we find them difficult to elicit, and, indeed, they can only be definitely elicited by re-enforcement. Here we have one of those curious cases with which one is now fairly familiar, in which there is very little doubt that we are dealing with a condition of tabes, but one which shows itself only in the optic symptoms, in a patient who, apart from the condition of his optic nerves, is practically healthy. The only other point which throws a certain amount of light on the condition which is probably present is the comparative difficulty in standing. You will see that there is a very slight knee-jerk even without re-enforcement, but it is distinctly increased on re-enforcement. The diminution in the knee-jerk is so great that it is almost as significant as if it were absent. His pupils react to light. And that you sometimes find in those cases of tabetic atrophy, viz. that in spite of the optic nerve being definitely affected the pupil reflex is still active. This is a type of a large class of cases of tabes, those which we speak of as tabetic optic atrophy, and they are distinguished by the fact that the optic atrophy is practically the only serious symptom which the patients have. Sometimes there may be some difficulty with the

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bladder, occasionally there are distinct lightning pains, but it is very rarely that there is definite ataxy present. So it is hardly correct to speak of such a case as one of "locomotor ataxy"; it is more correct to say it is a case of tabes dorsalis, of which the chief symptom is optic atrophy. The conjunction of the optic atrophy with the difficulty in eliciting the knee-jerks I think make it practically certain that the case is one of tabetic optic atrophy. There are no signs present to indicate the likelihood of the case being one of disseminated sclerosis; there is no bladder trouble and no tremor. There is nothing in the nature of an acute onset such as would suggest that it was an instance of post-neuritic atrophy, nor are the appearances of the discs at all compatible with such a view. The cases of tabes which one sees are roughly divided into two definite groups—one in which ataxy is present, and the other in which it is not present. The large class of patients in whom no ataxy is present are chiefly those in whom optic atrophy is the chief symptom. In many cases also in which gastric crises are present there is no definite ataxy, but in other cases, especially in which lightning pains are present and severe, and in which bladder trouble is present, and also ocular symptoms—that is to say, paralysis of ocular nerves—ataxy is quite a marked feature. There are other sub-divisions of cases of tabes, but roughly I think they do fall into those two distinct classes: cases which might be roughly classed as those in which ataxy is present (and which might be described as locomotor ataxy), and cases in which no ataxy is present, which are more correctly described as tabes, with perhaps gastric crises or with perhaps optic atrophy, or with possibly some other symptoms. So one must not be misled into hesitating to diagnose tabes if there is no ataxy present. And it points to the moral that the use of the name "locomotor ataxy" is rather to be deprecated, because ataxy in many cases of real tabes is not one of the symptoms present at all. With regard to the prognosis in this particular case the outlook is exceedingly bad. The chances are that, considering how much his sight has failed in the last eight months, the failure will go on until the sight is completely lost. And it is so in the great majority of cases of tabes with optic atrophy. They nearly always rapidly go on to such impairment of vision as practically amounts to blindness. And that forms a very distinct contrast with cases of disseminated sclerosis, because whereas it is the rule for patients with optic atrophy in tabes to become completely visionless, in cases of disseminated sclerosis it is very rare indeed to find a patient who is totally blind. Personally I have never seen a patient with disseminated sclerosis who was quite blind. I have seen one who lost the vision of one eye, but I have not seen such a patient completely blind from the optic atrophy of disseminated sclerosis. And that points to a distinct difference in the nature of optic atrophy in the two conditions. In tabes there is a gradual degeneration of the whole of the nerve-fibres, whereas in disseminated sclerosis there is a patch of inflammation in the optic nerve, possibly in one, sometimes in both, and according to the size of the patch of inflammation so you find the degree of optic atrophy, and according to the fibres of the nerve affected so you have the condition of the vision. The only drug which apparently does a little good in these cases is strychnine, and although it does not do very much in most cases it appears to lead occasionally to arrest of the process, and if arrest of it takes place one might expect a certain degree of improvement of vision to result from the subsidence of the inflammation, or from a diminution of the sclerotic condition which probably accompanies the optic atrophy.

Our next patient is a woman, and it is interesting to see such a case immediately after the one just seen, because here also I believe we have an anomalous case of tabes. She comes simply complaining of pains in the arms and legs, which she carefully describes. When you find a patient with pains such as she has—extremely severe shooting pain associated with cramp—you should be suspicious that the cases is one of tabes, especially if the distribution of the pain in the arms is such as she describes, namely in the ulnar distribution; for in most of the cases of tabes in which the upper limb is affected the ulnar portion of the limb is generally more affected than any other part, and is affected earlier. She has no difficulty in walking, but there is some trouble with the bladder. The three points which I should attach great importance to are, first of all these pains which she describes; and then the condition of the knee-jerks, the response of which on the two sides is very different, the

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knee-jerk being active on one side and inactive on the other; and thirdly the condition of the pupils. They are slightly unequal, but there is no reaction to light in them. So I think we have no difficulty, anomalous as the case is, in coming to the conclusion that we have here also a case of tabes. But it is a different class of case from that which we have just seen. It is one in which the pupil symptoms are present, whereas they are absent in the other case. There are no optic nerve symptoms, but the lightning pains are very severe. It is a contrast to the other case, and yet it resembles the other case in the fact that there is no ataxy present. So that in those cases of tabes one has to make the diagnosis on general principles, and if you find enough to justify the diagnosis you must make the diagnosis, no matter how unlikely it may seem on other grounds. A patient who comes complaining of what may be called rheumatic pains will be frequently found to be suffering from tabes. Another point is, how long do these pains last before definite symptoms come on? I have known a patient certainly suffer from lightning pains several years before any other symptoms had supervened at all. And as a rule you find when lightning pains are severe in the lower limbs that the knee-jerk is lost. In this case the knee-jerks are both present, but there is a marked inequality between them, the left knee-jerk being much more difficult to obtain than the right. So I think one would say, from those two cases, that tabes is not a disease which you can diagnose from the presence of any one particular symptom. Possibly the most certain definite sign of tabes is the Argyll-Robertson pupil, but that we know occurs in other conditions, and especially in general paralysis of the insane, and in the late stages of specific disease. The question of treatment is important in such a case because the presumption is that in an early stage there is no very severe affection of the cord itself. In such a case as this one we begin by giving iodide and mercury, or at all events by giving a course of iodide for six weeks or two months, beginning with ten grains three times a day and working up to twenty grains three times a day. With regard to the pains one has to treat them, and the lightning pains of tabes are successfully treated as a rule by such drugs as antipyrin or phenacetin, or phenacetin and caffeine, or sometimes aspirin. One has to find out in the case of each patient what is the most suitable. In the majority of cases phenacetin is as efficacious as any other drug. In some cases, on the other hand, in which phenacetin and antipyrin and the other drugs fail, one may find a very good effect from aspirin. But in the majority of cases phenacetin and caffeine together make an effective remedy for lightning pains, and one has no hesitation in giving that drug, to be taken occasionally, when the pains are coming on, for that will probably cut them short. And I think it is important in those cases to cut the pains short, because wherever there is pain going on the probable result of changes in the nerve roots, it is rather important to stop it, because no doubt the pain is associated with certain structural changes, and the more you can suppress those structural changes the better is the patient's chance of recovering from the disease, or, at all events, the greater is the chance of stopping its progress. So I shall order in this case iodide of potassium to be taken three times a day in ten-grain doses first of all, and then caffeine and phenacetin to be taken in powders or in tablets when the pains come on.

This man is sent up by his doctor with the complaint that he had an attack of loss of memory two years ago. He went home and did not remember any more for a day or two. He was not unconscious and has never lost consciousness. He says his memory is better sometimes than it is at others. He is a cab-driver, and was thrown off his cab two years ago. Up to that time he had been perfectly well. Physically he seems quite well and strong. There is no loss of power and no weakness in his limbs. He has had a sore on his leg ever since he was run over twenty years ago. He has two children, both of them healthy. The doctor gives a very curious history of this patient. He has been compelled to give up his work through absolute loss of memory. There has been no fit or unconsciousness, no convulsions, so far as one can make out, and after the accident he was brought to the doctor's house in a dazed condition, but not unconscious. He remained in this dazed condition for three weeks or a month, with very poor memory. For the last eight or nine years he has been a total abstainer. He has been a good servant, but has had to give up his work, because he cannot remember where he has

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to drive to, even for two minutes together. And one is asked to say what the condition is. In any case of that kind it is very difficult to be quite certain of a diagnosis. The probability is that in a case of sudden lesion of this kind the condition is one of vascular disease, because it still remains true that a sudden lesion is in all probability a vascular one, and if you get sudden impairment of memory, or sudden impairment of any mental function, with a dazed condition, it is very probable that some vascular lesion has taken place. And where there has been no absolute loss of consciousness the likelihood is that the lesion is one of thrombosis. In a case of this kind where will such a lesion be? It is not associated with paralysis of one or other side; there has never been unilateral weakness or any articulatory difficulty. So the lesion is almost certainly situated in such a region as is not associated closely with the motor functions, that is to say, it is almost certainly in the pre-frontal region of the brain. Such a condition of loss of memory is not usually isolated; there are certain other mental changes present as a rule as well, which it is not easy to recognise unless one is seeing them frequently. There may be loss of power of reading, and sometimes loss of memory for read articles, which is to be expected in any condition in which the memory is so much impaired as in this case. The important point in any such case is the prognosis. Here is a man, æt. 43 years, who is practically disabled from his work on account of this loss of memory, or at all events unable to follow the particular work which he has been trained for. The prognosis is bad as regards recovery of memory after three years. It is hardly likely that in a man aged forty-three there is going to be any very great improvement in the memory after a lapse of two years or more. So the outlook is one of some seriousness in that respect. As regards health and life generally of course the outlook is also a little doubtful, because when a man has had one vessel blocked, as we presume was the case here, it is possible that other vessels may follow suit, for the presumption is that atheromatous changes have taken place early in the vessels, and that this degeneration of vessels has not been restricted to one, but may affect other vessels. Such a condition in a man of this age is usually associated either with kidney conditions or with specific disease. There is no history in this case which tells of anything like syphilitic disease, and so far as I can find out there has never been any albumen present in his urine. So one is at a loss to account for this except by supposing that we are dealing with a case in which there are changes which have taken place in the vessels, possibly associated with cirrhosis of the kidney, but not necessarily so. In such cases one gets a little help from the ophthalmoscopic examination. The vessels have the "silver wire" appearance which is found associated with vascular degeneration elsewhere, and thus we are justified in looking upon such a view as I have expressed as the correct one. If one does not find any changes in the arteries, still I think there is no other way of accounting for such a condition except on some such hypothesis as I have stated. One likes usually to have some confirmatory evidence of such a view, and confirmatory evidence would be afforded by the presence of albuminuria, which is not present, or failing that the presence of recognisable changes in the arteries in the retina. One does not like to dogmatise too much about those changes, but I think you will see in an artery below the disc where it crosses a vein very definite evidence of the change to which I have alluded. It is a fibroid change in the arteries which is sometimes described as giving a silver-wire appearance. And that is a condition of degeneration which one would expect to find in such a case as this is. Among the numerous cases which we see here of arterial degeneration, it is curious how rarely one finds albuminuria present, and the same is true even of cases of albuminuric retinitis. We speak of such cases as albuminuric retinitis, and they are no doubt associated with kidney changes, but in many of them it is difficult on repeated examination to find any albumen in the urine. I remember an old lady, æt. 60 years, who was in the hospital some years ago with the most strlking albuminuric retinitis I have ever seen. She was an in-patient, and we examined her urine repeatedly, but were never able to find anything more abnormal than a low specific gravity, and there was never any albumen detected. But there were obvious vascular changes in the retinæ, and there was hypertrophy of the heart. And in this case the evidence of the vascular degeneration is found in the condition of retinal

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vessels, and in the presence of a condition which can only be due to some vascular lesion.

Here is a man who comes with this history: He sustained an accident on March 28th of this year when the head of his bicycle broke while he was riding. He was unconscious for three quarters of an hour, after which he only recollected seeing the policeman and the doctor. Afterwards he was unable to say who he was. He was not unconscious all the time. He had a fit and had to be shaken to rouse him. He has had fits since, and there "seemed to be a shade dropping over his eyes." As soon as he came to the ground he recovered consciousness. Sometimes this would happen twice or three times a day. He has had small pieces of bone coming away from his nostril, and some pus; he has a continual headache. He has never had anything the matter with the head before. He complains of having lost his sense of smell; he is restless at night and dreams a great deal. He has not been able to work since the occurrence of the accident. His wife says, "His frontal bone was loose for about a week after the accident." His nose was cut open by the fall. His last attack of unconsciousness happened about a fortnight ago, and he had had one on the previous Thursday. He has never had diplopia. It is rather an interesting condition because I should say it is probably a case of traumatic epilepsy. After his accident he probably had concussion, and ever since then he has not been quite the same as he was before. Apparently he has had sudden momentary attacks of loss of consciousness, and there has been a change in his mental condition, and especially he has suffered from a very great degree of irritability. Another interesting point is the condition of his nose and his loss of smell. Ever since the accident there has been some dead bone coming down his nostril. In such a case it is interesting to find out whether the loss of smell is of central or of peripheral origin, because in many cases of shock, such especially as results from bicycle accidents, there is a loss of smell, and in such cases apparently it is due to an actual tearing across of the olfactory fibres. But in this case I think it is more likely he has not had a fracture of his base, but that the loss of smell is probably associated with this local trouble in his nose. The future of a case of this kind is always somewhat uncertain. I think there is little doubt that those attacks are epileptic in character, and that means that his brain has been injured, *i. e.* there has been traumatism which has resulted in irritability of the cortex, and the evidence of that is the occurrence of *petit mal*. Although there has been some improvement with regard to the frequency of these attacks, still in his mental condition there has been no improvement, and his condition of irritability tends rather to increase than to diminish. So that the future of this case is complicated with the possibility of mental disturbance, and mental disturbance, after such an injury as he has had, is not uncommon. That is why I wished him to come into the hospital because it is impossible to appreciate the condition which such a patient suffers from unless he is under constant observation. Those attacks of momentary loss of consciousness are certainly, I think, epileptic. But they may be functional, because functional disturbance after such an accident is not uncommon. With regard to the condition of his nose, one would like to have that investigated, to see whether that loss of smell is associated with local disease or whether anything can be done to relieve this local condition of disease in the nose, which apparently results frequently in the discharge of dead bone and in the discharge of blood-clots.

Here is a male child, æt. 2 years and 3 months. He began to suffer from convulsive attacks eighteen months ago. He was acutely ill. Apparently he had been well in himself previously, and was born a healthy baby at full term. There was no difficulty about the labour. It was noticed that his eyes flickered when he was six weeks old; this flickering went away again. The convulsions affect the left side of his body, and they are more severe now than they were formerly, though they have not been quite so frequent during the last two days. The fits have always been left-sided. He has begun to speak and to stand by holding on to things, but his left leg is somewhat bent. He is said to be quite clean in his habits. The mother said she had a fall in the second month while carrying the baby. Sometimes he wets himself during an attack; unless there are several attacks practically continuously he does not go to sleep after them. He has had them as long as six hours at a time, passing from one into another. Now

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that the mother has left I may say that the outlook in this case is serious. The history is fairly clear, clearer than it is in many of these cases, because I think there is very little doubt it is a condition which has existed from birth. The mother says that at six weeks flickering of the eyes was witnessed, and you probably noticed just now while he was having the fit that the flickering of the eyes was a prominent feature, and that after the convulsion was over the eyes went on flickering. I have little doubt that this was the same sort of thing which was noticed when the child was six weeks old. But apparently eighteen months later there was a development of the attacks, that they became more severe. That is not uncommon in those cases in which some cortical irritation dates from birth. It is not unusual to have some slight sign of irritability early in life. A year or eighteen months later there are developments in the way of more severe symptoms of similar irritability. Now of course the attacks are very severe, epileptic in character, although I could not see, except in the eyes, any signs of clonic convulsion. The attack is almost completely restricted to the left side. So that we are dealing, in this case, with some actual local lesion. There is no doubt, I think, that the disturbance of the brain is on the right side of the brain almost entirely, and the probability is that it dates either from the time of birth or from intra-uterine life. First-born children are much more liable to such disturbances than are children born later—and this is her first child—because the difficulties in connection with the entrance of the first child into the world are very much greater, so that a large number of cases of birth-palsy are found in first-born children where usually there has been some considerable difficulty in connection with the labour and in which the condition has been such as to require instruments. As Gowers long ago pointed out, the reason for the occurrence of birth palsy in the cases in which there is instrumental labour is not because of the instruments, but because of the conditions which have made the use of instruments necessary, and in this case, although the obvious features of the condition are the fits which the child has, there is no doubt that these fits are associated with a condition of weakness of the left side. So it is a case of "birth-palsy," injury to the brain having occurred during birth or in intra-uterine life, and this injury has resulted in some weakness of the left side and the irritability which results in those frequent fits. The outlook for the child is exceedingly poor, both mentally and physically, because one must recognise it is almost certain to grow up with weakness of the left side; it is almost bound to develop into a case of hemiplegia in early life. And besides that, the condition which is present in the brain is almost certain to interfere with its development, so that it is likely that the child will grow up weak-minded. It is a very sad thing for the parents to have a child with this condition, but it is better for them to know the future of these cases than to live in a fool's paradise, for it is almost certain that the parents will have, at some future time, to take measures for the care of this child out of its own home. It will not be safe to place it in the company of other children, because such a child often develops a sense of irresponsibility and almost viciousness which is dangerous to others. The only drug which is known to help such a condition is bromide, and one gives it in order to prevent the irritability of the brain from increasing. Of course, recently there has been an attempt to deal with those cases by operation, and there is no doubt that if you recognise the condition early enough—that is to say, as soon as the child is born—you may conceivably deal with such a condition, especially if, as is probably the case, it has hæmorrhage over the vertex. In some cases of birth-palsy the result of hæmorrhage over the vertex—the hæmorrhage being associated with difficulties connected with the entrance of the child into the world—a clot has been taken away and the progress of the child after that has been done has been fairly satisfactory. But after the lapse of such a long time in this case nothing is to be hoped for by operation, because we are dealing with a case in which the injury has been to the brain, and the injury has interfered already with the brain in such a way and to such a degree that even if the child went on to develop well from this stage it is not likely to be quite like other children.

Here is a woman the subject of epilepsy. She is æt. 47 years. The attacks of epilepsy commencing at forty-seven years of age are not common, but still the disease may start at any time. There is a history of an accident at twenty-four. It may seem at first curious to attempt to

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connect an accident at twenty-four with fits at forty-seven; but she apparently fell down a trap, and was unconscious for some time afterwards, and it was only recently she heard of the unconsciousness which occurred after the fall. So there was apparently a considerable loss of memory after the fall down the trap. Whether she had a convulsive attack at that time as the result of the fall it is difficult to say. And sometimes one has to go particularly into the history of the accident, because in some cases it seems fairly clear that the so-called accident which began the attacks was only a fall which occurred in an attack. The first attack she knows of happened eight years ago. There is little doubt that patients often have attacks during the night long before any actual attack was known, and on investigating the matter one can sometimes find that some blood has been present or that there has been some disarrangement in the bed-clothes long before such an attack has occurred. It is not very common for epilepsy to occur for the first time at forty-seven years of age. It does occur at that period of life sometimes, but it is very doubtful whether the onset of epilepsy is associated with the onset of the menopause. Epileptic attacks may start at any age. I have known them start at seventy years of age and go on to ninety, so there is no particular reason why they should not start between forty and fifty years of age, and it is conceivable that in women the nervous phenomena associated with the menopause may make the onset of epilepsy easier. With regard to women, these attacks do not apparently come very often, and one's efforts are directed to getting the patient under the influence of bromide. I give bromide of sodium and nux vomica in these cases three times a day, and one must get the patient fairly under the sedative influence. The nux vomica does not counteract the power of the bromide, and it prevents the depression which the bromide alone is apt to produce.

*July 13th*, 1908.

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We have received from Mr. H. K. Lewis, Publisher, 136, Gower Street, a catalogue of 'Lewis's Circulating Library' revised to the end of 1907. The price is 5*s*. net., but to subscribers it is 2*s*. net. The book is an unrivalled list of modern medical and scientific text-books and special monographs.

## A CLINICAL LECTURE

ON

# CARCINOMA OF THE ASCENDING COLON.

Delivered at the Post-Graduate College, West London Hospital.

By LEONARD A. BIDWELL, F.R.C.S.,

Surgeon to the Hospital and Dean of the College.

I WISH this evening to claim your attention to the consideration of a case of carcinoma of the ascending colon which was in the hospital some months ago. We will then compare this case with cases of carcinoma in other parts of the large intestine. The history of the case was as follows:

Mrs. R—, a well-nourished woman, æt. 40 years, consulted my friend Dr. Penny at the commencement of July last year on account of a severe attack of pain just below the ribs. She had always had perfect health, was very active, and was in business. She had no irregularity of her bowels, but she had a curious fad that it was a cleanly thing to wash out her colon about twice a week. She used to administer a simple enema of about half a gallon of water. Eighteen months ago she had a slight attack of pain just below the ribs, which, however, did not last long. It came on after eating currants, and when she washed out her colon she found many currant seeds in the washings. She had about three other similar attacks each time after eating fruit with seeds. These attacks were not, however, of sufficient importance to induce her to see a doctor. She said her bowels were regular, her motions formed, and no blood or mucus was passed with them. She had never been jaundiced after these attacks. She regained her perfect health immediately the pain passed off, and maintained it till July 3rd, 1907, when she was seized with more severe pain just below the ribs on the right side; this was accompanied by retching, but she did not vomit, and the pain lasted four or five hours. Since then she had always more or less pain beneath the costal arch, and the pain went through to the back. The bowels had been quite regular since. Now these symptoms were strongly suggestive of gall-stones, and this was the diagnosis which was

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made when I was asked to see her with a view to operation.

I saw her on July 24th, and I found her well-nourished, not anæmic, and without any sign of jaundice. On examination the abdomen was slightly full but not distended. There was a well-marked tender spot just below the eighth right costal cartilage corresponding to Mayo Robson's point for gall-stones, but no tumour corresponding to an enlarged gall-bladder could be made out. A well-marked tumour could be felt in the right loin which was evidently a floating kidney, but this was quite painless. On further examination a small movable tumour, the size of a bantam's egg could be felt just about McBurney's point. It was slightly tender. It was movable in every direction, but downward traction gave rise to pain. There was no enlargement of the liver, and nothing abnormal was discovered by either rectal or vaginal examination. There was no ballooning of the rectum.

Now it was quite evident that this small tumour was the thing which was giving trouble, so we might consider what a tumour in this position could be. First it might be (1) an inflamed and thickened appendix, and the attack of acute pain might have been an acute appendicitis; (2) an inflamed displaced ovary or tube, but an inflamed ovary or tube in this situation would not be so mobile; (3) an enlarged mesenteric gland secondary to appendicitis or to an ulcer of the cæcum (this latter is not a rare condition); (4) a tumour of the pylorus; I have discovered the pylorus in this situation in a case of great dilatation of the stomach; and (5) a cancer of the small intestine, or cæcum.

Now, I quickly narrowed my choice between a subacutely inflamed appendix and a carcinoma of the lower part of the small intestine. The history of the present attack would do equally well for either disease, but the previous history of abdominal pain after taking fruit with seeds was much more suggestive of some constriction of the lumen of the small intestine, so I gave as my diagnosis that the case was probably one of carcinoma of the small intestine or cæcum, but that it might be only an infiltrated appendix. As the patient was in continual pain I sent her into the hospital at once, and I arranged to operate on the following day.

At the operation a carcinoma of the cæcum and ascending colon was found and was removed.

I want to discuss with you first the symptoms and then the operation undertaken.

In the first place we must recognise that the symptoms of carcinoma of the cæcum and ascending colon are very indefinite in the early stages, and the growth has usually existed for a considerable time before any suspicion of its presence arises. It is easy to understand why the symptoms are so much less acute and so much longer delayed than is the case with a growth situated in the lower part of the bowel, since in the cæcum the intestinal contents are liquid, and so are enabled to pass through a very narrow lumen, and unless some undigested portion of food such as a grape skin or pea is impacted in the growth, a lumen the size of a lead pencil will be amply sufficient for the passage of the intestinal contents. Again, as the intestinal contents are fluid they do not stretch and irritate the surface of the growth in the same way as do the solid contents of the sigmoid, and so there is seldom bleeding or the passage of mucus. The disease may exist for from one to two years before it gives much inconvenience, and its early symptoms may be *nil*. Again, the narrowing is very gradual and this gives time for the small intestine to undergo hypertrophy of its muscular coats, so as to gain additional strength to drive the fluid contents through the constriction; this exactly resembles the hypertrophy of the bladder seen in old-standing stricture of the urethra (in this case we found considerable thickening of the small intestine). So for a long time the only symptom of this serious disease will be some slight griping pains in the lower part of the abdomen, and these usually are not severe enough to cause the patient to seek medical advice, or if they do seek advice the symptoms do not appear sufficiently severe to cause the doctor to make a careful abdominal examination. Such pains are most marked a little time after food. It is important to remember that there will probably not be constipation, and that the motions will be formed and of a natural size, since the absorption of fluids and the shape of the motion is determined in the sigmoid, and the peristaltic contraction of the whole of the transverse and descending colons gives a sufficient *vis-à-tergo* to empty the rectum. I have already stated that bleeding is

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not so common in growths in this position owing to the fluid character of the intestinal contents, and so melæna is not a usual symptom, but when it does occur it will be in the form of completely altered tarry blood, resembling that seen after duodenal hæmorrhage, and not the clots and bright blood seen in carcinoma of the sigmoid. Sometimes, however, melæna may be an important, and, in fact, the only symptom, as in a case which I saw with my friend Dr. Reckitt, of Boston, last year. This patient was a lady, æt. 60 years, who had enjoyed good health till Christmas, 1906, when she began to get feeble without any apparent cause, and during the summer of 1907 she became intensely anæmic and weak. She had no obstruction, but the motions were very dark in colour. There was a well-marked tumour in the right iliac region, and there was some distension of the coils of the small intestine. As the patient was clearly bleeding to death from the tumour an operation was advised. I removed the tumour, which involved the cæcum and the ascending colon, and I made an anastomosis between the ileum and the transverse colon. On opening the tumour the growth was found to be ulcerated and fibrinous blood-clot was adherent to it. This was a case where hæmorrhage and not obstruction was threatening life.

If we have a case which has given trouble neither by colicky pain nor by hæmorrhage, we shall get no symptoms till acute obstruction occurs, and this usually occurs in consequence of the plugging of the opening by means of some undigested matter. In one case of acute obstruction that I treated I found a mass of grape skins in the cæcum just below the growth. Now in these cases, as the blocking is effected suddenly, the symptoms are very acute. Pain is severe, and in thin patients peristaltic waves are very evident; vomiting is an early symptom and very quickly becomes feculent. The amount of fæcal matter vomited is sometimes very large, and after it has been evacuated the patient often feels much better and takes food again, but of course the vomiting recurs after a short interval. Naturally the condition most likely to be confused with this is the obstruction caused by a strangulated femoral hernia, and I need hardly warn you of the necessity of searching the femoral rings before considering any other cause of obstruction; the hernia may be very small (sometimes no bigger than a damson plum), and its presence is seldom alluded to by the patient, so it may be missed by the most competent surgeon. The symptoms of acute obstruction by a growth are, nowever, never quite so acute as those caused by a strangulated hernia, as in the former case the obstruction is entirely mechanical and the intestines have become accustomed to some interference with their movements, while in strangulated hernia there is always some irritation of the mesentery, and in addition there may be thrombosis of the veins and gangrene.

The main point, however, in the diagnosis of a carcinoma of the cæcum is the presence of a tumour, and except in the very stout a tumour will always be felt so soon as the slightest symptoms are complained of; it is much more easy to feel than a tumour, say, in the sigmoid, which is situated in the false pelvis. The really important point to realise is the comparatively slight symptoms which are associated with a growth, so that we shall not hesitate to make a diagnosis of cancer when a tumour is discovered in the right iliac fossa.

Before leaving the question of diagnosis I should like to briefly refer to the symptoms of cancer in other parts of the colon. Some of you saw me operate on a case of carcinoma of the transverse colon. Here the history was very similar, except that the pain was of a rather longer duration, and there was marked vomiting owing to the proximity of the growth to the stomach. The emaciation also was more pronounced. The lumen of the gut was greatly contracted and I have never seen such intense distension of the cæcum, nor such extreme hypertrophy of its walls. The obstruction had been rendered acute by kinking of the colon just behind the growth produced by traction of the omentum which was adherent in a left inguinal hernial sac. There was no absolute obstruction since the bowels acted after a simple aperient. On rectal examination there was no ballooning of the rectum. There had been neither melæna nor mucus. In this case after removal of the growth the ends of the transverse colon were united by end-to-end anastomosis.

In a case of growth of the descending colon on which I operated lately the history was as follows: Some discomfort and irregularity of the bowels for a few months, and then obstinate constipation about six weeks before admission, which, however, yielded to a purge; this was followed by diarrhœa

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which had persisted. Here the growth was irritated by semi-fluid fæces, causing the secretion of a good deal of mucus, which produced the diarrhœa. In this case, too, on rectal examination we found well-marked ballooning of the rectum—a valuable sign of any obstruction in the lower part of the colon or sigmoid. With the sigmoidoscope we should have seen the growth easily, but unfortunately it was out of order when required. In the case of a growth a little lower down, say at the middle of the sigmoid, we get more mucus and blood in the motions and very often scybala. Obstructive symptoms appear early and are not acute, vomiting being quite a late symptom, the most common symptoms being mucous discharge and blood and then alternating constipation and diarrhœa. Another point is the extreme fœtor of the stools, this being probably due to decomposed blood. In addition we have alteration in the shape of the motions and often piles. We nearly always notice emaciation and anæmia.

In the lower part of the sigmoid no tumour can be felt in the abdomen, but it can often be felt *per rectum* and the sigmoidoscope shows it at once. Here as well as in the rectum the symptoms which cause suspicion are alternating constipation and diarrhœa, or persistent diarrhœa, melæna, mucus in the motions, and offensive discharges. Cases with such symptoms, in middle age, should always be examined with a sigmoidoscope.

We must now leave the question of diagnosis and pass on to that of treatment.

Wherever possible the growth must be removed, but it is not advisable to unite the divided ends of the ascending or descending colon as these parts have such an incomplete peritoneal investment that union is not likely to be complete. It is therefore advisable in all such cases to close the cut ends and to restore the continuity of the intestine by a lateral anastomosis between the ileum and colon.

In my early cases I divided the operation into two stages, and at the first operation did a lateral anastomosis between the ileum and transverse colon; about fourteen days later I removed the growth and closed the divided ends of the ileum and ascending colon. This proceeding was open to two objections: First, some patients experienced such great relief by the ileo-colostomy that they refused to submit to the radical operation, and secondly, the growth was several times found to be more adherent at the second operation and its removal was more difficult than it would have been at the first operation. I now, therefore, would only recommend the operation to be done in two stages in very feeble patients or in those who collapse under the operation, and I also strongly advise that the growth should be removed at the first operation, and if after removal of the growth the patient is too feeble to stand the closure of the divided ends and lateral anastomosis a Paul's tube should be tied into each end of gut and the wound closed, a lateral anastomosis being done a fortnight later.

The *operation* is performed by making an incision about four inches long along the outer edge of the right rectus muscle, dividing the anterior layer of the sheath pushing the muscle inwards and then dividing the posterior layer of the sheath and opening the peritoneum. The peritoneum is then divided on the outer side of the cæcum and ascending colon where there is little vascularity; this is done about half an inch from the gut, and then the ascending colon and the tumour can be brought easily out of the wound. The anterior layer of the meso-cæcum and meso-colon is then divided about half an inch from the bowel and reflected back towards the root of the mesentery. The ileum is then clamped about two inches from the ileo-cæcal valve, and a clamp is applied to the transverse colon about an inch above the growth. The bowel is ligatured on the growth side of each clamp to prevent escape of contents; the bowel is then divided, and each end of gut is surrounded by a sterilised mop to prevent any fouling of the wound. The meso-cæcum and meso-colon are then clamped in section and divided, when the tumour will be freed from all its attachments.

The sections of the mesentery are then ligatured, and if the patient is able to stand the complete operation the ends of the ileum and of the ascending colon are then occluded; this is done by passing a purse-string suture round each end and then invaginating the tied ends by Lembert's sutures. A lateral anastomosis is now done between the ileum about three inches above its divided end and the transverse colon. Some surgeons implant the cut end of the ileum into the colon, but this method is more likely to be followed by contraction of the new opening than the method of lateral anastomosis.

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The ileum should be united to the colon in such a way that the peristaltic waves in the two portions of bowel are in the same direction.

After completing the anastomosis the edges of the peritoneum on each side of the cavity from which the cæcum has been removed must be brought together carefully. In order to provide drainage a stab puncture is then made in the loin just below the first rib, and a drainage-tube is introduced to lie just below the peritoneum. This is essential, as it is nearly impossible to prevent soiling of the wound when removing the cæcum. The anterior abdominal wound is completely closed.

If the condition of the patient has prevented the completion of the operation as described, Paul's tubes are tied in the cut ends of the bowel, the posterior parietal peritoneal edges are brought together and a stab drain is made in the loin. In order to close the artificial anus it will be best to perform an ileo-sigmoidostomy, since the abdominal incision required for the operation will be far removed from the artificial anus. Before commencing the operation the artificial anus should be plugged with gauze and its edges united with silkworm-gut sutures and the whole of the old wound covered with gauze and sealed with collodion. The new incision is then made in the outer edge of the left rectus and the sigmoid exposed. The ileum is then brought up into the wound and united laterally to the sigmoid; after this the ileum is completely divided between the anastomosis and the artificial anus. Without the division of the ileum fæces would still be discharged from the artificial anus. Nothing need be done to the free end of the ascending colon as this will contract and only a small mucous fistula will be left. It is interesting to find that after these operations of ileo-sigmoidostomy the patient does not suffer from diarrhœa, since the absorption of fluids is very rapid in the sigmoid and rectum.

With regard to prognosis in these cases the immediate results of the operation are very good; the mortality does not exceed 10 per cent., and if cases of obstruction are excluded the mortality should not be much more than 5 per cent. There is a danger of secondary growths developing in the liver, but local recurrence is rare, since the mesenteric glands are freely removed. There is little risk of any subsequent obstruction, even if the growth recurs in the liver, so the remainder of the patient's life is rendered comfortable.

One thing is quite certain, that the earlier the operation is performed the less is the risk to life and the greater the probability of a permanent cure.

*July 13th, 1908.*

# ON SOME MODERN METHODS OF INFANT FEEDING.*

By FREDERICK LANGMEAD, M.D., M.R.C.P.,
Physician to Out-patients, Paddington Green Children's Hospital; Medical Registrar, St. Mary's Hospital.

ALL who see infants professionally, whether in private or hospital practice, must be impressed by the many examples of children who are suffering from grave mistakes in diet. Instances of over- or under-feeding, feeding irregularly and with foods physiologically incomplete or positively harmful, are before our eyes every day. There are many causes which contribute to this effect; poverty, vice, and neglect all play their part, but undoubtedly the most important factor is ignorance. Often the mother and her relations know nothing about the subject, and they come to rely on authority. Too often they find it, for so it seems to them, on walls and hoardings, or in magazines, particularly of the quasi-religious sort, expressed in terse telling sentences, illustrated by all the joys of country life, or horrors reminiscent of the Indian Famine. Sometimes they put their faith in their medical attendant. Let us look to it that they come not to lean upon a broken reed.

Any lecture on the artificial feeding of infants would be incomplete which did not recognise its limitations. There is no perfect substitute for breast milk. Modify cow's milk in what manner we will, we still fall far short of attaining an ideal food. Bottle life is artificial life for the baby, and like catheter life is fraught with many dangers. Whoever takes a child from the breast during the first six months of life incurs a real responsibility, and should only do so after careful investigation.

Frequently, however, the mother either cannot or will not nurse the baby at the breast, and we are then compelled to find a substitute.

In England wet nursing is generally disliked and is not readily obtainable, and we must perforce use the milk of other animals.

(1) *Ass's milk.*—That of the ass is often, somewhat uncharitably, stated to resemble human milk most closely, but if we look at the following

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comparative table we see that there are wide differences:

| | Ass's milk (Ellenberger's [1] analysis). Per cent. | Human milk. Per cent. |
|---|---|---|
| Water | | 87·60 |
| Proteid | 1·2–1·7 | 1·52 |
| Fat | 0·1–1·7 | 3·28 |
| Sugar | 5–6 | 6·50 |
| Mineral matter | 0·3–0·4 | ·27 |

The chief point of resemblance lies in the proportion of proteid present, for in contrast to other milks it is usually even smaller than that of human milk. This no doubt increases its digestibility, because the firmness of the curd which is produced in the stomach depends, among other circumstances, upon the amount of proteid present, but it has two great disadvantages: It contains a very small proportion of fat, only about 1·5 per cent. as compared to 3·2 per cent. in human milk—a very serious deficiency in an important ingredient, and secondly, it is expensive, and even if entirely satisfactory could only be obtained by the few.

(2) *Goat's milk.*—The latter objection applies, too, to goat's milk, and, moreover, the chief chemical disadvantages of cows' milks are found even more pronouncedly in that of the goat. There is nothing to be gained by using the less readily procurable milks. Cow's milk will be our substitute except under unusual circumstances. Let us examine very carefully the chief differences between it and human milk.

(3) *Cow's milk.*—With the differences in chemical composition you are, doubtless, well aware. They may be represented in the following table, which represents the average results of a great number of observations (Hutchison, [2]).

*Chemical Differences.*

| | Human Milk. Per cent. | Cow's Milk. Per cent. |
|---|---|---|
| Water | 87 –88 | 87 –88 |
| Proteid | 1 – 2 | 3 –4 |
| Fat | 3 – 4 | $3\frac{1}{2}$ –$4\frac{1}{2}$ |
| Sugar | 6 – 7 | 4 –5 |
| Mineral matter | 0·1– 0·2 | 0·7 |
| Reaction | Alkaline | Acid |

It recalls at once to our minds that although the total amount of solids is equal in the two milks, the proportions differ. Cow's milk is richer in proteid and in mineral matter chiefly, but also to a slight extent in fat. Human milk is sweeter from a larger percentage of sugar.

But this by no means enumerates all the distinctions, for the constituents differ very greatly qualitatively as well as quantitatively.

The nitrogen in milk is partly present in the form of unknown bodies, which we call extractives, and these bear a larger proportion to the total volume in human than in cow's milk; in the former they constitute $\frac{1}{4}$th of the total volume, in the latter $\frac{1}{16}$th. The proteid, too, is different, for whilst in human milk it is composed of equal parts of casein and lactalbumin, in cow's milk there are six parts of casein to one of albumen; and albumen is much the more digestible constituent. Herein there lies an important difference, from the point of view of the relative digestibility of the two milks.

*Fat.*—The fat of human milk is in a finer state of emulsion than in cow's milk and contains a higher proportion of oleic acid—a fatty acid having a low melting-point. Both these differences again favour a higher digestibility of human milk.

Lastly, *the mineral salts* are also dissimilar not only in their total amounts but also in their combination. We have seen that calcium and phosphorus are present in greater amount in cow's milk. There is this difference also: in human milk the phosphorus is almost entirely organically combined; in cow's milk, less than half is in this form. It is present in human milk in the more easily assimilable form. So that wherever we find qualitative differences between the various constituents of human and cow's milk, they are in favour of human milk considered as an infant's food. It is these qualitative distinctions which make modified cow's milk, at best, but an imperfect substitute.

We now come to the practical inference from these fundamental chemical facts. The question we have to settle is, How can we best modify cow's milk to make, if not an ideal, at least a not unworthy substitute for human milk? There is no doubt that the chief difficulty which the infant encounters in digesting cow's milk is due to the coarseness of the curd. If we examine the vomit or the stools of a baby with whom cow's milk is disagreeing, we see masses of stringy undigested curd. This is just as we should expect, for the curd is formed by casein, and casein is in considerable excess in cow's milk.

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But there is another reason, and one which until comparatively recently has not been accredited with sufficient importance. I refer to the excess of calcium salts in cow's milk. Bunge gives the proportion of lime salts in human milk as ·03 per cent., in cow's milk as no less than ·17 per cent. Now Arthus and Pagés long ago showed that if cow's milk were treated with oxalates and fluorides—substances which precipitated the lime salts—it could no longer be clotted with rennet, whilst if lime salts were added the clot became denser. Remembering this experiment we cannot be surprised at the indigestible curd of cow's milk.

It is this coarse curd which we must set ourselves to prevent.

*Dilution.*—The first method is that which has been most in vogue for many years, viz. *dilution* By adding water we decrease the amount of casein and of calcium, and indirectly the size of the curd.

This is, no doubt, a method of great value, but I think we are somewhat too satisfied with it, for when we dilute the casein and the calcium we also dilute the other constituents of the milk—the albumen, fat, sugar, extractives, and phosphorus—some of which we know full well cannot be spared, and others there is very good ground for believing are also of considerable value.

The following table shows approximately the percentage of the most important constituents in various dilutions:

| | Human milk. | 1 part of cow's milk to 2 parts of water. | Equal parts. | 2 parts of cow's milk to 1 part of water. |
|---|---|---|---|---|
| Proteids | 6·5 | 1·0 | 1·75 | 2·0 |
| Fats | 3·5 | 1·3 | 2·0 | 2·6 |
| Carbohydrates | 6·5 | 1·5 | 2·75 | 3·0 |

Supposing ℥vj of milk diluted with an equal quantity of water, are being given, about two teaspoonfuls of centrifugalised cream and two teaspoonfuls of sugar require to be added, and even then the percentage of proteid is slightly too high. This need for adding more cream to diluted milk is a very real drawback, especially among the poorer classes. To give diluted milk to which cream has not been added is to endanger the infant's growth and health by rickets, and yet we know that to order cream to be given in the case of a large proportion of the population is but mere waste of words, for they cannot afford it. We see the truth of this contention daily among the children of the poor, for we cannot get away from the fact that many cases of rickets arise after feeding with diluted cow's milk, and I have seen very severe examples among infants who have, as far as one could tell, been fed strictly according to the tables distributed by certain institutions. To return to the question of the density of the curd. It is true that this is not so large as in undiluted milk, for if we halve the casein we shall halve the amount of curd; but it must be remembered that the proportion of casein to lime salts remains the same, and so we shall obtain curd having similar characteristics. To make it looser dilution with *barley-water* is recommended. Now barley-water is made usually in two ways, either boiling water is poured on to pearl barley and the fluid is quickly strained off, in which case it is still practically boiled water and offers no special advantages or disadvantages; or the barley or barley-flour is first soaked in water, which is afterwards brought to the boil and kept just simmering for about half an hour. If prepared by the latter method it contains an appreciable amount of starch (¾ per cent.), which endows it with both bad and good qualities; for although the starch makes the fluid more viscous and thus prevents the shrinking and hardening of the curd, yet starch is a substance which the infant's digestive powers are not fitted to deal with. Personally I think the disadvantages outweigh the advantages, and prefer to dilute with plain boiled water.

*Humanised milk.*—Another device which is used to fit cow's milk for infant feeding, is that by which *humanised milks* are prepared—milks diluted with an equal part of water and centrifugalised. The upper half of the milk is then withdrawn. It contains practically all the fat of the milk, but only half of the other ingredients. Sugar is subsequently added, sufficient in amount to attain the correct percentage. Such a milk is still too rich in calcium salts, and the curd is still of the same type. It is practically a commercial imitation of the home methods for the modification of cow's milk which have been in general use. It is necessarily dearer than ordinary cow's milk.

The disadvantages of diluted milk are these:

(1) They are a bulky form of food given at an age when any distension of the stomach is particulary undesirable.

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(2) More manipulation is required than may be safely entrusted to many mothers.

(3) The dilution necessary at special months and the subsequent variation of amounts of added constituents is too involved to be accurately complied with by the average mother.

(4) The curd, although small in bulk, remains still much more coarse or less flocculent than that of human milk.

(5) Cream must be added, but costs more than many mothers can afford, and usually contains preservatives.

*Whole milk feeding.*—With these disadvantages of feeding with diluted milk before us, it is well to consider whether it is possible to modify milk in such a way that it may be given whole. By so doing we shall be giving an excess of proteids and mineral matter, and consequently must find some method of altering these constituents that the curd found in the stomach is no longer the solid massive curd which is produced by unmodified milk.

The method which is largely used on the Continent is that of *sterilisation.*

This procedure was first advocated by Budin (3), who has now for many years fed infants under his care, in Paris, on sterilised undiluted milk. At his consultations for nurslings, where the mothers attend regularly to receive their supplies of sterilised milk, and where the babies are regularly weighed, he has had unusual opportunities of observing the value of this method. He is very emphatic in his opinion. He says he has *never* seen bad results from its use.

He writes: "I carefully refrain from asserting that during the first months of life milk ought invariably to be prescribed unmixed with water. Just as infants may not be able to support their mother's milk if it is too rich, so they may not tolerate some specimens of cow's milk overcharged with butter or casein. Cow's milk is not of constant composition; it varies with the breed of the animal, its feeding and other factors. Again, infants may or may not be healthy, and in the latter case their digestive tubes have need of judicious treatment. Thus, each infant must be dieted according to existing conditions, and it may be of service to add a variable quantity of ordinary water, barley-water, lime-water, or other diluent. My custom is to prescribe pure milk, and any departure from this is determined on only after careful observation."

His method of sterilisation is by keeping the milk for forty-five minutes at a temperature of 100° C. This is best done in some form of apparatus, the most familiar variety of which is that devised by Soxhlet.

The reason for the greater digestibility of sterilised milk over unmodified cow's milk has been carefully investigated by Chavane ('Du Lait Sterilisé,' Paris, 1893), who finds "that the clot of milk thus sterilised offers no resistance or sense of elasticity to the finger on pressure, but yields like a thick fluid. And on microscopic examination, although the fat globules are not altered appreciably, the particles of casein are found to be smaller and more homogeneous. Evidently this sterilisation below boiling point modifies the casein so as to affect the state of subdivision of the clot and also the molecular structure of the particles." Professor Budin (4) says after experience with whole milk feeding since 1892, that a tumid abdomen is almost unknown among his nurslings, that he is unacquainted with milk dyspepsia, that no single case of rickets has occurred, and that he is still looking for his first case of infantile scurvy. Such a result could not be said with truth about feeding with diluted milk.

Variot (5), Comby (6), Lasard (7), and many other French authorities (8–11) corroborate his statements. There is, however, one objection to this method as applied to the poor. They could not be entrusted to efficiently sterilise. To carry it out properly it would be necessary to establish similar consultations for infants, or *Gouttes de lait* as they are called, in England, so that at present we must find some other method for general use.

*Citration.*—By sterilisation we modify the *casein* of cow's milk; we have not yet considered whether it is possible to alter the other cause of the greater density of the curd, viz. *the excess of calcium salts.* This has been shown to be possible by Sir Almroth Wright (12), to whose work the next form of modification is chiefly due. As far back as 1893 he showed that there are two kinds of curdling of milk: (1) curdling with rennet when the curd is firm; (2) curdling with acid when it is loose. Curdling with rennet is that form which accounts for the indigestibility of cow's milk. Remembering the work of Arthus and Pagés, who, as I have

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mentioned, precipitated the calcium salts by the addition of oxalates and chlorides, and so prevented rennet curdling, he suggested that sodium citrate, a perfectly harmless salt, might be used instead of the poisons employed by the two physiologists. He himself suggested that whole milk should be used.

Dr. Poynton (13) brought this method more prominently before the profession as recently as 1904, but combined it with dilution, though to a less extent than is usually practised. Many authors have written on this subject since his paper, and, indeed, this treatment has now become very general, and is used in a large number of the children's hospitals in London and elsewhere. But with very few exceptions all who have hitherto employed citration have also followed Dr. Poynton and combined it with dilution. This I hold to be unnecessary except during the first two weeks of life. The excess of proteid is simply passed *per rectum* and gives rise to no gastro-intestinal disturbance in my experience, and if undiluted we have the advantage of keeping the milk in as small bulk as possible, and with a proper percentage of fat.

For more than twelve months I have employed citration without dilution for my out-patients at the Paddington Green Children's Hospital, and am more than gratified by the results. The exact method of procedure I employ is as follows: When the baby is first brought to the hospital, supposing it is a case which is not thriving on artificial feeding, it is weighed and its weight is recorded on a chart which has been devised for the purpose; the number of feeds *per diem* and the amount of each feed are then determined upon. No hard and fast line can be drawn with regard to either the frequency or size of each meal, since so much depends upon the size and vigour of each particular child and on its previous feeding, but the following rule affords a useful standard: For the first five weeks, as far as possible, meals are prescribed every two hours, for the second five weeks every two and a half hours, and afterwards until the sixth month, every three hours. This, of course, applies to day feeding only. By night, *i. e.* from 10 p.m. to 7 a.m., two meals are given during the first three weeks, one during the second three weeks, and afterwards the meals are slightly rearranged so that the baby goes without the bottle from 11 p.m. to 5 a.m.:

*By day:*

| | 1–5 weeks. | 5–10 weeks. | 10 weeks to 6 months. |
|---|---|---|---|
| *Feeds:* | 2 hourly | 2½ hourly | 3 hourly |

*By night* (10 *p.m. to* 7 *a.m.*):

| 1–3 weeks. | 3–6 weeks. |
|---|---|
| 2 meals | 1 meal |

For quantities I use the following table, which is taken from Holt (14) and slightly modified:

| | Feeding quantities. | | | Approximately. |
|---|---|---|---|---|
| 1st week | ℥j | –℥iss | = | ℥j |
| 2nd–3rd week | ℥iss | –℥iij | = | ℥ij |
| 4th–5th ,, | ℥iiss | –℥iiiss | = | ℥iij |
| 6th–12th ,, | ℥iij | –℥ivss | = | ℥iv |
| 3rd–5th month | ℥iv | –℥vss | = | ℥v |
| 5th–9th ,, | ℥vss | –℥vij | = | ℥vj |
| 9th–12th ,, | ℥viiss | –℥ix | = | ℥viij |

This table errs somewhat on the side of liberality when dealing with undiluted milk, and so I always start somewhat below the standard here given, for gastro-intestinal disturbances are the result not of too little but of too much food. Of necessity there are many cases of infants far below the normal weight, and with poor powers of digestion, for whom a meal far smaller than the average is essential.

I then prescribe for each ounce of milk ordered two grains of sodium citrate. This is given in the form of a watery solution, ℥j of which has to be put into each feed. For example, if 4 oz. of milk are given every three hours, the prescription is thus:

| | | |
|---|---|---|
| Sod. citr. . . | gr. viij | |
| Aq. ad. . . . | ℥j | 3 hourly. |

The mother is instructed to bring the milk to the boil and then while it is cooling to add one teaspoonful to each feed. Sodium citrate is very soluble in water, so that it is possible, no matter how many ounces of milk are being given at a feed, to dissolve the citrate in ℥j of water.

At subsequent attendances the child is weighed again, at first weekly, afterwards fortnightly or every three weeks, and its weight duly recorded. The amount of milk is adjusted to the progress of the child, which is estimated by the general condition and the evidence of the scales. At about five months of age citration is gradually diminished until the advent of teeth permits of unaltered milk and a more extensive dietary. If weak citrate solution is kept in a warm place a fungus is apt to grow on the surface, but this is obviated by adding a few drops of chloroform water.

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This method is so simple that the mother can not easily make a mistake in preparing the milk, which is a great advantage. It requires very little manipulation, and without doubt the less manipulation milk undergoes the better for the baby. It produces in the stomach a fine flocculent curd, which infants can deal with who have been screaming constantly, drawing their legs up and showing other evidences of indigestion after diluted milk. There is no danger of rickets, for it is only the excess of calcium which is thrown down, which, even then, is still given in the milk. Moreover, Dingwall Fordyce (15) has fed rats on citrated milk and no rickets has resulted. There is no danger of scurvy.

You will notice that the mother is instructed to bring the milk to the boil; this does not appreciably alter its taste, neither does it sterilise it, but it renders it free from living pathogenic organisms. When we remember that in a report issued by Dr. Collingridge (16), Medical Officer of Health for the City of London, that of 39 samples of milk representing the supply to London from 22 counties, 7·9 per cent. were infected with the tubercle bacillus, 17·9 per cent. with the *Staphylococcus aureus*, and 25·5 per cent. with Gaertner's bacillus, the necessity for at least bringing the milk to the boil is impressed upon us. Also the curd with citration is finer in boiled than in unboiled milk.

### *Results.*

I have now watched several of these patients from early infancy until dentition, some of whom weighed far below the average when they were first seen, and am able to say that they become strong, well-developed infants, and that rickets does not result.

Constipation is said to be the most troublesome effect, but I have been unable to satisfy myself that it is more in evidence in babies fed on citrated than on other forms of modified milk. The stools are more bulky, but in those patients in whom constipation has been serious it was so before they came under treatment.

Some fifty cases have been treated in this way at the Paddington Green Children's Hospital. They were all children who were wasting under other methods of feeding. In any case in which the mother's statement that the child was getting thinner seemed to want confirming two weighings were first taken, the first on the child's first attendance, the second after a week's interval. Many were marasmic, two or three weighing about 5 lbs. at ten weeks to two months of age. The previous feeding had been by the breast, by diluted cow's milk, and by many kinds of artificial foods. One was fed entirely on albumen-water as a last resort; three, according to their mothers' statements, were brought because they had been given up by their doctors, and in one instance a medical certificate of death had already been promised.

Some have been lost sight of, as always happens in hospital practice, but with the exception of two or three who attended for about one week only all have gained steadily in weight, many making up for lost time and reaching the normal before the citration was replaced by unaltered milk.

Time, no doubt, will show us more clearly the limitations of the usefulness of whole citrated milk for infant feeding, but at present I regard it as the most satisfactory method we possess.

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*July 13th, 1908.*

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# THE CLINICAL JOURNAL,

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## A CLINICAL LECTURE

ON

# A CASE OF HÆMOPHILIA.

Delivered at St. Bartholomew's Hospital.


By D'ARCY POWER, M.A., M.B.Oxon., F.R.C.S.Eng.,

Surgeon to and Lecturer on Surgery at St. Bartholomew's Hospital; Consulting Surgeon to the Bolingbroke Hospital and to the Victoria Hospital for Children, Chelsea.


GENTLEMEN,—The subject of hæmophilia is interesting and is well worthy of thought because it leads to such disastrous results, and we know so little of its cause that we are powerless to cope with it. I make no apology, therefore, for bringing this boy before you because he has been under my own observation, at intervals, for the last thirteen years, and I am thus able to give you a first-hand account of his condition from childhood.

The patient, as you see him, is a well-grown boy, æt. 19 years, with a muddy complexion made worse by numerous acne spots. He walks badly because the movement in both knees is impaired and apparently more so on the right than on the left side. His left elbow, too, is somewhat bent and the knuckle of the right index finger is markedly swollen, whilst the whole finger is reddened, swollen and glossy.

When I first saw him, he was admitted under my care at the Victoria Hospital for Children, on September 2nd, 1895—nearly thirteen years ago. He was then seven years old and he had fallen downstairs five days previously. He was brought to the hospital on the day after the accident on account of a swollen knee which was diagnosed as an acute synovitis and was thought to be the result of the accident. The joint was hot, swollen and painful, and as he did not improve under treatment he was admitted to the hospital.

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At this time he was a pallid and sickly-looking boy with a temperature of 101° F. The left knee was tensely distended and was kept slightly bent. Free fluctuation could be obtained from the swelling. The joint was not tender and pain was only complained of at night. There was some evidence of bruising.

The joint was kept at rest whilst ice and evaporating lotions were applied, but no suspicion of the true nature of the case seems to have been aroused, for on September 4th a hydrocele trocar and cannula were inserted into the joint for the purpose of drawing off the synovial fluid. Instead of the clear synovial fluid, which was evidently expected, dark treacly blood appeared, but in no great quantity because it was too thick to pass along the cannula. There was a considerable oozing of blood from this puncture throughout the night, but the swelling of the joint was said to be less on the following day.

The patient remained ill and with a raised temperature, which attained a maximum of 103° F. on September 17th, but as he had improved a little he was sent to the convalescent home at Broadstairs on September 25th.

He was re-admitted to the hospital on October 16th, 1895, stating that he had been sent back from Broadstairs a week before owing to an outbreak of scarlet fever in the home. His mother brought him on account of the constant oozing of blood which had followed the removal of two fragments of decayed deciduous incisors. His left elbow was also swollen, the result, as she thought, of some accident a few days before. I found him blanched with loss of blood and with a small, rapid pulse. The gum of the upper jaw was still bleeding at the points where the teeth had been extracted. There was a thick spongy clot of blood in each little cavity. The right elbow-joint was distended and showed signs of bruising. There was marked absence of pain or tenderness on moving it. The left knee was still slightly swollen, but it was not painful.

The knee was put up in a plaster-of-Paris case, the sockets of the teeth were plugged with gauze soaked in tincture of hamamelis, and drachm doses of the tincture were ordered every three hours. The boy was sent home on the following day.

Four days later the boy's mother brought him to the hospital again saying that the bleeding from his gums stopped after three days, but that both his elbows were now swollen.

The patient was again sent to the convalescent home at Broadstairs, but in three weeks' time he returned with an immense subcutaneous extravasation beginning in the left popliteal space and extending as low as the left ankle. The left knee was also more swollen. The leg was allowed to rest on a pillow for three days, and was kept cool with a light gauze dressing soaked in lead lotion. It was then covered with a thick layer of Gamgee tissue over which a plaster-of-Paris bandage was applied.

The general condition of the patient caused me a great deal of anxiety at this time. He was extremely anæmic; he was drowsy and he was very apathetic. The pulse was feeble and rapid, and the temperature again rose steadily until it reached 103° F. Physical examination showed the heart to be natural, and there was nothing abnormal to be detected in the liver or the spleen. The boy improved quickly with careful nursing, and it is noted on November 29th—nine days later—that he sits up, takes his food and is interested in his surroundings. A small superficial abscess formed in the left groin on December 2nd. The abscess was not connected with the lymphatic glands. It was allowed to open itself and it was healed on December 9th. At this time the left knee moved fairly well and was painless; the calf of the left leg was much reduced in size, though the skin covering it was still deeply pigmented, the ankle was normal and the knee was nearly straight. The right leg, including the joints, appeared to be unaffected.

The patient was re-admitted to the hospital in January, 1896, on account of a swollen right elbow, the skin over the upper part of the ulna appearing bruised.

Nothing more was seen of the boy for more than a year, when he was re-admitted to the hospital on April 20th, 1897. He was now nine years old, and he had bruised his face by falling upon it ten days previously. The bruising was followed by great swelling, which extended all over one side of the face and then began to subside.

On admission the boy was found to have much swelling of the cheek and lips on the left side, with some ecchymosis of the left conjunctiva. The skin over the centre of the tumour was hot and thinned.

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A new house-surgeon who knew nothing of the former history, and who evidently was not able to inquire about it, mistook this swelling for an abscess and made an incision into it. No pus escaped, he notes, but only dark blood in a fluid condition. The boy pulled off his bandages during the night, and lost much blood. I saw him next day, and ordered an injection of citrate of ergotine, $\frac{1}{100}$th gr.; he was also given 15 gr. of calcium chloride every three hours. The bleeding stopped and in a few days the boy was discharged.

He was admitted again to this hospital with a cut on his lip, which had been bleeding all the previous day. It had stopped bleeding at five o'clock, the day before his admission, but the hæmorrhage began again early next morning. The bleeding was stopped by the application of Paquelin's cautery at a dull red heat, a layer of Whitehead's varnish being afterwards applied. The patient's right knee had been swollen for a fortnight.

The boy's mother gave the following history:

The patient had always been liable to swelling of the joints since he was two years old, and he had on that account been treated for rheumatism. The attacks of swelling were preceded by much pain, restlessness at night, and a peculiar increase in the size of the small blood-vessels on the cheeks. The latter phenomenon she had noticed herself, and she volunteered the information about it without being questioned. She had also noticed before she brought him to the hospital for the first time that "when he cuts himself he usually bleeds for a week."

A younger brother, æt. 14 months, in 1895 bruised his knees and elbows by crawling. In January, 1896, he suffered three times from severe bleeding at the nose, which left him very weak, and he died of bleeding at the age of eleven years after three teeth had been extracted.

The boy's father shows no tendency to bleed; the paternal grandfather died of rheumatic gout.

The mother shows no tendency to bleed, but one of her sisters died young of "bleeding from the head"; another of the mother's sisters died of phthisis; the maternal grandfather died of influenza at the age of seventy.

This case presents many points of interest both to the physician and to the surgeon. As a surgeon, I turn first to the state of the joints. The right knee and the knuckle of the right index finger are in a different condition from the left knee and the left elbow, and the joints on the right side were affected later than those on the left, as we know from the history.

The right knee at the present time is enlarged into a curious globular shape, which is quite different from the enlargement caused by tubercle, syphilis, or rheumatism. There is no wasting of the muscles partly because there is no trophic disturbance, and partly because the joint has not been painful, and it has not, therefore, been spared in walking. The skin over it is natural in colour and it is not hotter than the left knee. The synovial membrane is clearly thickened and the cavity of the joint contains rather more fluid than there should be if it were healthy. There is no creaking on movement, and the enlargement appears to be chiefly in the bones themselves, for the condyles of the femur are larger than natural and their edges as well as those forming the head of the tibia are markedly "lipped."

The joint cannot be fully straightened, and the boy limps in walking.

The right index finger seems to be in a still earlier stage of inflammation, for it is swollen and tender, the joint surfaces are not lipped, but the surrounding fibrous tissues are thickened.

The left knee and the left elbow are in a more advanced state of disease. They are wasted, there is no appreciable increase in their synovial membranes; the lipping is not universal at the edges of the bones, but is aggregated into nodular masses, which appear to be true bone, and their movements are very considerably impaired, with marked creaking as though the cartilages had undergone destructive changes.

I thought it would be interesting to see what appearances these joints would show by radiography, and I therefore obtained skiagrams of them. You will see that the knees show very little change and that any "lipping" must be due to a multiplication of the cartilage cells and not to any calcification or ossification, for there is no trace of any change in the skiagraphs. The elbow, on the other hand, shows marked evidence of "lipping," more especially round the head of the radius (see Fig.). I have hopes, therefore, that the patient

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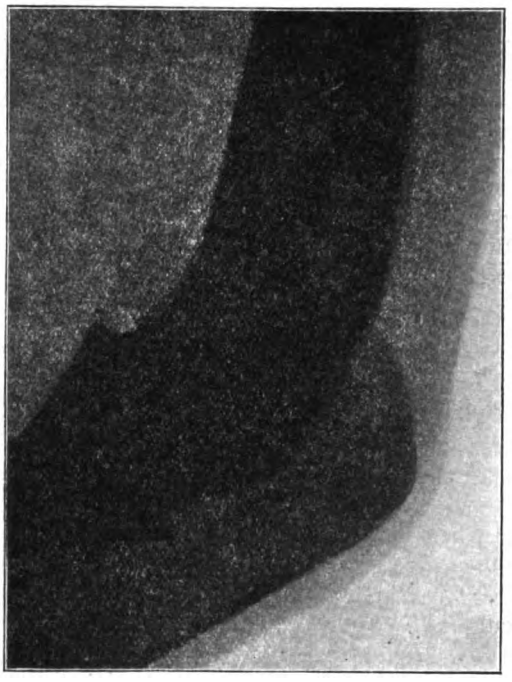
will obtain greater use in his legs, though it is impossible to promise him much improvement in his elbow.

Our museum affords us some interesting specimens showing the nature of the changes which take place in hæmophilic joints. We owe a debt of gratitude to Dr. Wickham Legg not only for the specimens, but for the care with which he has recorded the histories and the symptoms of the patients from whom they were obtained.

The first series of joints were obtained from a boy, æt. 9 years, who died in the hospital under the care of (Sir) Thomas Smith on October 26th,



1881. It is said that he was sitting on a doorstep on October 10th when a drunken woman fell upon him and made him bite his lip. He continued to bleed from this slight wound in spite of all the resources of the hospital until he died sixteen days later.

Both the parents of this patient were healthy. There was no history of bleeding on the father's side and the mother knew none among the older members of her family. She had eight children, and five were living when the patient was in the hospital. She had one miscarriage. One of her children, a boy, æt. 4 years, died twelve months previously of an injury to the back. He was subject to bleeding from the nose lasting sometimes two or three days, and on one occasion he bled on and off for three weeks.

The boy from whom the joints were obtained was also subject to bleeding from the nose, often for two days without ceasing, and he also bled very much from any small wound, such as a cut finger. When he was one year and nine months old he suffered from a swollen right knee. He was treated for it in Charing Cross Hospital for five months and it remained somewhat swollen until the time of his death. When he was three years old he fell down and cut his lip. The hæmorrhage was so severe and prolonged that he was taken to King's College Hospital, where he remained a month. A similar accident happened to him six months later, and he was again admitted as an in-patient and was kept for a fortnight.

On arriving at the age of six years his ankle suddenly became swollen, and he was taken to King's College Hospital where an incision was made into it. The bleeding from the wound could not be stopped for a week. Eighteen months before his death he was a patient in Darker Ward of this hospital with an injury to his ankle, and twelve months later he was re-admitted because he had nearly cut off the top of one of his thumbs, the hæmorrhage could not be stopped for a fortnight.

The mother has a third son who does not bleed.

The post-mortem examination showed the existence of much extravasated blood in the right popliteal space which invaded even the substance of the muscles bounding the space.

The knee was slightly flexed. The patella was fixed to the front of the femur by an adhesion that was broken through without difficulty. A superficial gap was then seen in the cartilage on the femur where the patella had been fixed to it. The cartilage on the patella had been absorbed and fibrous tissue occupied its place.

The synovial membrane was stained of a yellow-brown colour. The ends of the bone at the knee, where they were not covered by cartilage, were coloured in like manner. The cartilage preserved its usual appearance, and a similar condition was found in the left ankle-joint, but there was no erosion of the cartilages. There was very slight staining also of the right ankle; the other joints were natural.

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The second set of joints are equally instructive. They show more advanced changes and are probably more comparable with the condition of the joints in the patient whose case we are studying. We are again indebted to Dr. Wickham Legg for the specimens as well as for the history of the case.

They were obtained from a boy, æt. 13 years, who was admitted into the hospital under the care of Mr. Langton on January 22nd, 1883. The notes record that his nose began to bleed on January 21st. He vomited a bowlful of blood at 6.30 p.m. soon after his admission on January 22nd. He was much collapsed, and the anterior and posterior nares were plugged by the house-surgeon. He passed a quiet night, but on the following morning his temperature was 101·4° F., and in the evening 100·6° F.

There was still some oozing from the left nostril on the 24th. He was very drowsy, and slept all day. The morning temperature was 102·4° F., but in the evening it had fallen to 99·2° F. The plugs were withdrawn on January 25th, and fresh plugs steeped in tinct. ferri perchlor. were substituted. Nutrient suppositories were given three times a day. The patient was delirious in the evening. The temperature in the morning was 102·4° F. and in the evening 102·6° F.

The plugs were removed on January 26th, and the nostrils were syringed out with iced water. Some drops of blood came from the right inner canthus. There was no blood in the urine. The left ankle-joint was swollen, and there was a large ecchymosis in the groin. There was also much puffy swelling of the face. The temperature at 10 a.m. was 105·4° F. He became delirious towards evening, when the temperature rose to 106·4° F., and he died at 8.40 p.m.

When the boy was three years old he bled for a long time from a small wound on the tongue. At the age of six he bled for eight weeks after a tooth had been extracted. He bruised easily, and had frequently had painful swellings of the joints.

One brother died of epistaxis at the age of three years and a half, but these are the only instances of bleeding known in the family.

The *post-mortem* examination showed an extravasation of blood as large as the palm of the hand. It had occurred in the subcutaneous tissue of the outer part of the left groin.

Both knees and the left ankle-joint were examined. Fresh dark blood with a small amount of clot was found in the ankle without any structural change in the joint; the cartilage was pink from post-mortem staining.

No recent blood was found in the left knee, but its traces were seen in the deep russet-brown colour of the synovial membrane. The cartilages preserve their pearly-white aspect. At the under surface of the external condyle of the femur where it meets the pressure of the tibia the articular cartilage is worn thin and granular over a space of half an inch in diameter. The ligaments of the joint were unaltered.

The destructive changes had advanced still further in the right knee. The connective tissues of the joint were also stained a deep brown colour.

The articular cartilage was wanting over the points of pressure, and bone thinly covered by cartilage has developed at the periphery of the joint. The central part of the articular cartilage at the under surface of the condyles of the femur is thin, worn, and rough. It is fissured in various directions and is laminated. It has lost its close attachment to the bones so that a knife can be passed beneath it here and there for a distance of 4–6 millimetres. The edges of the partially detached cartilage, when raised, can be seen to be ragged, fibrous, and split into layers like that of a joint affected by chronic rheumatic arthritis. There is a prominent lip of bone round the edge of each condyle; this lip is somewhat nodular and is thinly covered by cartilage.

The same description holds good for the articular surfaces of the tibia and patella. There is a gap in the cartilage of the external condyle of the femur, which is situated on the front surface just at the line of division between the articular surfaces. It extends to the bone, and is widest at the outer part. The edges of the cartilage bounding the gap are smooth and rounded.

A microscopical examination of the cartilage made by Mr. Bowlby showed that the hyaline matrix was undergoing fibroid degeneration, and that there was a multiplication of the cartilage cells with disintegration of their capsules.

By the light of these specimens and with the help of the skiagrams we can obtain an accurate picture of the condition of this boy's joint. The bones are thinned from disuse, for the skiagrams show a great excess of cancellous tissue. The car-

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tilages have undergone extensive disintegration due to increased proliferation of the cartilage cells, the cells having become disintegrated on the articular surfaces of the joint, whilst they are heaped up at the edges where they are protected from pressure and are covered by the reflexion of the synovial membrane. The synovial membrane is thickened by inflammatory changes, and is discoloured by the repeated hæmorrhages which have taken place in its substance. A few fibrous adhesions derived from the changes associated with the organisation of the extravasated blood limit the movements of the joints and make the boy a cripple. Broadly, therefore, the patient is suffering from chronic and multiple arthritis with some fibrous ankylosis. The stress of the inflammation has fallen upon the articular cartilages of the affected joints, to a less extent upon the synovial membrane, hardly at all upon the bones.

The boy is interesting in many ways as a living example of a most deadly disease. His history is singularly complete, and he is here, fortunately, to corroborate my statements. There is no question as to the diagnosis. He is suffering from hæmophilia, not, perhaps, in its most severe form, and it seems certain that as he grows older the attacks are becoming less frequent and certainly less severe. He recognises this himself, and attributes it to the fact that increase of years brings with it increase of wisdom, and he becomes more and more careful to avoid unnecessary risks.

The attacks have been of the usual character: external bleedings, which have been caused by very slight injuries, spontaneous hæmorrhages into the connective-tissue planes, and painful hæmorrhages into the joints. He has been spared the terrible attacks of nose-bleeding which are often so marked a feature in hæmophilia. But he has run many risks, some of them quite unnecessary, as when a zealous house-surgeon punctured his knee thinking he had an acute synovitis, and when another laid open the hæmatoma on his face, believing that the swelling was suppurating. The only excuse to be offered is that the boy had a high temperature and was extremely ill. But their mistakes should make you remember that a rise of temperature usually takes place during the hæmophilic attacks, and that extreme listlessness is sometimes a characteristic feature of the disease.

The patient belongs to the more rare class of hæmophilics whose attacks are preceded by storm signals, for you remember I told you that his mother used to foretell an attack by noticing the heightened colour in his cheeks. These prodromal symptoms vary in different patients, though they all point to increased blood-pressure. They occur three or four days before the actual bleeding takes place, and they are usually physical, though in some cases they are psychical, as when the child is said to be more cheerful, or to have increased acuity of sight or hearing.

The onset in the two children of this family did not differ from that which is usually observed. The first attacks were noticed in this boy soon after he was twelve months old, whilst in his brother, who died of epistaxis, there was bruising whilst he was learning to crawl. Hæmophilia may, however, show itself much earlier, for children have been born with hæmorrhages, and it has long been recognised amongst the Jews and other Semitic races who practice ritual circumcision. It seems, therefore, that the tendency to bleed is truly congenital, though it does not show itself in very young children, because they are protected from injury. It thus happens that the earliest signs are seen as soon as the child is left more to its own devices, and consequently to the usual chances of injury; but against this must be set the fact that the separation of the umbilical cord is hardly ever attended with serious bleeding, even in those who afterwards prove themselves to be pronounced bleeders.

Absolutely nothing is known of the pathology or cause of hæmophilia. It was once thought to be racial, and that the fair northern races were especially liable to it. But it has long been known amongst the Jews and Arabs, whilst every year information is forthcoming about its existence in parts of the world where it had previously passed unrecognised. We are driven to think, therefore, that it occurs amongst all races, and that most is known about it where the medical men have been most observant and best educated. Indeed, this very boy shows how easily such a case might have been overlooked if he had not happened to have been admitted into a hospital where we are naturally on the look-out for unusual cases. If he had lived in a country district at a distance from medical aid, the attacks might have passed unrecorded, and if he had come to us in his present state and without a good history, we should probably have thought

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that he was the subject of some unusual form of chronic rheumatic arthritis.

The family history of hæmophilia shows that in many cases it is an inherited disease, and the ancestral history is well put by Dr. Wickham Legg in the following words: "The disease does not seem to descend from father to son, nor through the sons of a bleeder family, free from the disease, to their offspring. As a general rule the sons of bleeders and the sons of the brothers of a bleeder, who are free from hæmophilia, show no signs of the disease. But the daughters of a bleeder, like his sisters, pass on the disease to their male offspring," so that this boy's nephews might show the disease in an aggravated form. The disease, too, is known to occur in every class of the community, neither poverty nor wealth appearing to act as a predisposing cause.

Too much, however, must not be made of the question of heredity. Many people are entirely ignorant of the medical history of their family, and in many cases hæmophilia seems to occur sporadically. The family history is by no means clear in the present instance, although two boys are hæmophilic. The inheritance came as usual, if the mother's story is to be believed, through her side of the family, because one of her sisters died of bleeding. But hæmophilia in a woman is extremely uncommon. Indeed, if too much stress is laid upon the necessity of a family history in hæmophilia, it will often be difficult to make an accurate diagnosis, and a hæmophilic joint will be looked upon as either tuberculous, rheumatic, or gouty.

The differential diagnosis of hæmophilic joints is often a matter of considerable difficulty. During the acute stage the inflammation may be considered an acute traumatic arthritis, simple or suppurating, or a rheumatic synovitis; in the later stages it may be mistaken for tuberculous inflammation, when it occurs in children, for gout or osteo-arthritis in older persons.

The mistake may be disastrous when it leads to operative measures, for the surgeon may find himself unable to control the hæmorrhage he has occasioned. It was made twice on this patient, as I have already told you, but he has survived. It is very easy to mistake a hæmophilic joint for tuberculous arthritis because both occur in children, both begin with an ill-defined history of injury and both affect the knee and the elbow more often than other joints. The distinction is made by the bruising. It is, I think, very rare to find an ecchymosis in tuberculous arthritis, and the appearance of bruising in or near a joint which is being treated for this disease should certainly set the surgeon thinking and should make him inquire more closely into the history of the case. Mistakes may be avoided if it be borne in mind that the attacks of hæmophilia are intermittent and that they are always associated with a rise of temperature which lasts during the attack.

The older writers on hæmophilia looked upon the swollen joints as evidence of a gouty diathesis, and believed that the disease was a gouty inheritance, but more exact knowledge has disproved this assumption. The early age at which the joint affection appears and the history of the case enables a ready distinction to be made between gouty arthritis and hæmophilic inflammation of the joints as well as between hæmophilia and osteo-arthritis, though there is at least one form of infective arthritis which affects young persons and runs a chronic course, leading to the destruction of cartilage, the lipping of the bone and the hypertrophy of synovial membrane which characterise the chronic joint affections grouped under the general heading of osteo-arthritis.

Nothing is known about the cause of hæmophilia. Attempts have been made to explain it upon anatomical and upon physiological grounds, but with equal want of success, for explanations founded on facts discovered in one set of cases have been upset by the absence of these factors in other series where the bleeding was equally troublesome and dangerous.

The popular idea attributes hæmophilia to the fact that the patients are deficient in a skin, and Professor Virchow to a certain extent approved this theory when he asserted that it was due to an abnormal thinness and narrowness of the blood-vessels which allowed the blood to escape owing to the disproportion between the amount of blood in the body and the size of the vessels. This view was afterwards assisted by assuming that hæmophilic patients were the subjects of an intermittent "hydræmic plethora." They were therefore sweated and given diuretics, but without any good result. Professor von Recklinghausen considered hæmophilia to be a disorder of the nervous system, an hypothesis which was adopted by Henoch, who

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looked upon it as a paralytic dilatation of the smaller blood-vessels allowing increased diapedesis.

The physiological explanations depend on the fact that the coagulation time of the blood is unduly lengthened, and Sir Lauder Brunton advanced the theory that it might be due to a deficiency in the lime salts, a suggestion which was accepted and acted upon by Sir Almroth E. Wright. The hypothesis commends itself to my mind if it be coupled with some intermittent failure of the vaso-motor centres in the neighbourhood of the fourth ventricle, for in this patient, at any rate, an initial constrictor influence was followed by a subsequeut vaso-dilation. But as I have already said it is useless to argue from a single case. So far as is known—and very careful examinations have been made—there is no morphological change in the heart, blood-vessels or blood itself to account for hæmophilia.

The treatment is unsatisfactory because so little is known of the cause. When a patient is known to be a bleeder prevention is better than cure. The mother must be made fully aware of the dangerous condition of the child, and she must be told in great detail how the bleeding may be caused and of its danger, for it is extraordinary how stupid people are, and how quickly they forget the warnings which have been given to them. Small wounds and trivial cuts are as dangerous to hæmophilic patients as more serious injuries. The drawing of teeth is, perhaps, the most common starting-point of a fatal hæmorrhage, for the epistaxis which runs it close as a cause of death often begins without any known cause. Hæmaturia and melæna are sometimes symptoms of an attack, and the mother should therefore be told of the possibility of their occurrence.

During the active hæmorrhage the patient must be treated in such a way as to cause him the least possible disturbance. The child should be put to bed in a cool and darkened room, with only so much clothing as will prevent him taking cold. The mechanical means employed to stop the bleeding should be such as do not cause the subsequent formation of eschars. The thermo-cautery and perchloride of iron are unsatisfactory for this reason. I prefer to cleanse the bleeding part as gently and thoroughly as possible with a stream of hot salt solution and then pack the nostril or the tooth socket with sterile gauze, and if the packing has to be repeated, as is generally the case, soaking it in hazeline, Ruspini's styptic, a saturated solution of antipyrin, or some preparation of adrenalin, hemisine being perhaps the most convenient. It happens only too frequently that all local measures prove useless, and the patient continues to bleed until he is reduced to the lowest ebb, when he takes a turn and revives. When this has happened, but not until then, rectal injections of saline solution prove beneficial, and he should be treated like a patient recovering from extreme shock. During the stage of active bleeding large doses of calcium chloride may be given at short intervals, but they must not be continued for any length of time because the earlier effects of the drug shorten the coagulation time of the blood, but if it be continued for too long the coagulation time is increased. I therefore give half-drachm doses every two hours for six hours, persevering if any results seem to be attained, but giving it up if the bleeding still continues.

When the bleeding is taking place into the joints the patient should be kept in bed and the swollen joint should be kept at rest upon a splint, whilst calcium chloride is given by the mouth. When the joint has ceased to be painful and the temperature of the patient has fallen to normal, passive movement and massage are likely to yield a more useful joint than the older methods of keeping the limb at rest and encased in a plaster-of-Paris bandage. Every effort must be made to promote the absorption of the blood effused into the joint, for the subsequent degenerative changes in the joint seem to be due to the irritation caused by the presence of the blood. In the later stages of joint disease associated with hæmophilia the use of massage and of hot-air baths after the manner recommended by Tallerman afford some relief, and in this particular patient I have been employing horse serum in 10 c.c. doses in the hope that the general condition of the patient as well as the state of his joints might be improved.

*July 20th*, 1908.

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FRIEDMANN says that only typical psychoses of pregnancy are considered as an indication for the induction of an abortion. He thinks that a more important indication for this procedure is found in the "psychopathic reaction" observed in some individuals, which results in an exaggerated and morbid fear of childbirth.—*Med. Rec.*, vol. lxxiv, No. 1.

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# ACUTE ABDOMINAL LESIONS OF PELVIC ORIGIN.*

By THOMAS G. STEVENS, M.D., B.S.Lond., F.R.C.S.Eng., M.R.C.P.Lond.,

Physician, Hospital for Women, Soho Square; Physician to Out-patients, Queen Charlotte's Lying-in Hospital; Obstetric Tutor, St. Mary's Hospital.

CASES of acute and sudden illness due to lesions of pelvic origin come before us sufficiently often to make it clear how difficult their differential diagnosis may be, and how important is their appropriate treatment. The patient's life sometimes hangs upon a correct diagnosis being made and appropriate treatment being carried out, and the amount of time at our disposal for clinical observation of the patient is often very short. To realise the truth of this we have only to think of some cases of acute hæmorrhage from rupture of an ectopic gestation or of peritonitis from the rupture of some pelvic pus collection. In either of these instances delay of a few hours in some cases makes all the difference between life and death. The difficulty of diagnosis arises because the symptoms belonging to most acute pelvic lesions in women are the same, and secondly, because the only person who can give any history as a rule is the patient, who is in no condition to be able to help us in that direction. If we run over in our minds the common acute pelvic lesions in women, we find that the symptoms common to them are: (1) Abdominal pain and rigidity of the abdominal muscles; (2) collapse; (3) vomiting, more or less; (4) rise of temperature and pulse; (5) anæmia. It is true the last belongs particularly to cases of hæmorrhage, but it may be present in other conditions, and in a purely general way we must admit that these symptoms occur more or less in all. If a definite history of the case be obtained it will always assist us in making a diagnosis, but as histories are often very imperfect, especially if the patient is unconscious or very ill, we must in many instances make our diagnosis on signs and symptoms alone.

Let me recall to you the common conditions which give rise to acute abdominal symptoms of pelvic origin. They are:

(1) Intra-peritoneal hæmorrhage caused by rupture of an extra-uterine gestation, tubal abortion, or rupture of a vein in the broad ligament or in connection with a tumour; into a Graafian follicle which may have burst; after interstitial hæmorrhage into the ovary.

(2) Axial rotation of an ovarian tumour with torsion of its pedicle; axial rotation of a tubal swelling.

(3) Rupture of an ovarian cyst.

(4) Rupture of a pyosalpinx.

(5) Peritonitis, local or general, due to gonorrhœa or other infections.

In this list cases of acute illness following labour or abortion, such as rupture of the uterus and puerperal sepsis, have been purposely left out because their causation is clear even if their diagnosis is sometimes difficult, and they form a group by themselves outside the scope of this paper. Taking a broad view of the above-mentioned conditions we find that they can be roughly separated into groups by means of their salient features. Thus if we consider the cases in which definite tumours exist we may classify them thus:

(*a*) A large tumour rising above the pelvic brim is present, usually an ovarian cyst with a twisted pedicle.

(*b*) A small tumour not rising above the pelvic brim is found. It may be an ectopic gestation, a pyosalpinx, the remains of a ruptured ovarian cyst.

(*c*) An indefinite swelling is found, continuous with the uterus and fixing it, and may be due to local pelvic peritonitis, acute salpingo-oöphoritis, or hæmorrhage into the broad ligament.

(*d*) No tumour at all can be felt. Pelvic peritonitis or intra-peritoneal hæmorrhage from a ruptured vessel.

If, on the other hand, we consider symptoms only, we find that in all cases of acute progressive hæmorrhage there is a rapid increase in the pulse-rate without any rise of temperature, progressive anæmia and pallor and the other general signs of serious internal bleeding. Whilst in inflammatory conditions we find rise in temperature as well as in the pulse-rate and no progressive pallor, whilst a lesion may be found with which inflammatory symptoms are bound up and leucocytosis may be present. Such classifications as these do not help

* A Lecture delivered at the Polyclinic.

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us much in an obscure case, but they give us the broad distinction upon which diagnoses depend, and bearing them in mind whilst investigating a case we may be able instinctively to assign the lesion to one of them.

To consider now these various conditions in detail and taking *intra-peritoneal hæmorrhage* first, we are struck at once with the fact that it is only progressive hæmorrhage which stands out as self-evident. It can hardly be conceived that a diagnosis will not be self-evident when we see a patient who has been apparently in good health suddenly seized with violent abdominal pain accompanied by a progressively rising pulse and progressive pallor. If at the same time we find the abdomen tender to the touch and a small lump in one posterior quarter of the pelvis the presence of a ruptured extra-uterine gestation is almost a certainty. Rupture of a vein in the broad ligament, or of a vessel upon a tumour, such as a fibroid, have been known to cause progressive hæmorrhage, but they are so rare as to be almost negligible events. But if these progressive cases are self-evident, it is equally true that they form but a small percentage of the hæmorrhage due to tubal gestations, and that cases in which the hæmorrhage is small at first are the important ones to diagnose early, that by appropriate treatment a possible large fatal bleeding may be averted. The usual history of a case of tubal gestation includes as a rule one or more attacks of acute pelvic pain before a large bleeding occurs. These are often attended by passing faintness, and may be sufficiently serious to call for medical aid. These attacks we know now are due to hæmorrhage around the ovum and into the wall of the tube, small in amount, but producing great pain on account of the tension they produce in the tubal sac. These small hæmorrhages may precede either rupture of the sac or extrusion of the ovum from the fimbriated extremity—a condition known as tubal abortion. When the latter occurs it practically never produces progressive hæmorrhage, although the collapse induced at the time of its occurrence may be great. It is essentially rupture of the tube which produces the great hæmorrhages. It need hardly be said that if a diagnosis can be made in these conditions of small hæmorrhages around the ovum, an immediate operation will be attended by but little risk and the best prospects of cure. In a classical case the history is as follows: One or two menstrual periods may have been missed, often with a long interval since the birth of a child. Other signs of pregnancy such as morning sickness may be present and breast signs may appear. Then come one or more attacks of acute pelvic pain, accompanied by a blood-stained discharge from the uterus and followed by an acute intra-peritoneal hæmorrhage. If all cases gave such a history we should not make many mistaken diagnoses, but unfortunately there may be no period of amenorrhœa, there may have been no previous conceptions, and the very first attack of pain may be the one accompanied by great internal hæmorrhage. The physical examination before a great hæmorrhage has occurred may reveal very little, especially if the patient is tender. In such an event it is most important to examine under an anæsthetic, for in that way only can the enlarged tube be discovered. It is by no means uncommon at the first onset of hæmorrhage to make out nothing but some fixation of the uterus, and to be unable to feel any tubal enlargement. Under an anæsthetic it is not likely that this will be overlooked, and at all events there will be a much greater chance of making a diagnosis. The same remark applies to cases of tubal abortion, in which the amount of bleeding is seldom very great.

Bleeding from the uterus, which is an important accompaniment of tubal gestation, usually occurs when a slight hæmorrhage has occurred in the tube, converting the gestation sac into a mole. It is an important symptom and signifies the breaking up and discharge of the decidua, which always forms in the uterus itself in these cases. Where the history and signs of ectopic gestation are clear, this bleeding is an important corroboration, but if the case is at all doubtful it may prove a stumbling block, and perhaps suggest to us that we have to deal with a simple uterine abortion. In fact many cases of tubal abortion and slight bleeding from ruptures of the tube are diagnosed as uterine abortions, and their true nature is not discovered until later. In some instances the discharge of a complete cast from the uterus may assist us in diagnosing the case. An examination of this cast shows it to consist of decidual cells exactly like those of a normal pregnancy, but it shows no trace of a decidua reflexa (now called capsularis), and shows, of course,

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no chorionic villi. The finding of chorionic villi naturally implies a uterine pregnancy, but it must not be forgotten that cases of combined intra- and extra-uterine pregnancy have occurred.

The signs of rupture of a vessel in the broad ligament or in connection with a tumour will always be those of severe hæmorrhage, and, except in the case of the latter, can hardly be distinguished from those of ruptured tubal gestation with great hæmorrhage.

Hæmorrhage into a Graafian follicle and secondary bursting into the peritoneum has been known to produce fatal bleeding, and in the same way interstitial hæmorrhage into the ovary has given rise to serious symptoms. Such lesions will hardly admit of accurate diagnosis, and will probably be mistaken for ruptured gestation sacs. This mistake is not a bad one if it leads to prompt and appropriate treatment. The bad mistake to make is to overlook the possibility of hæmorrhage and treat the case on expectant lines.

In the differential diagnosis of all hæmorrhage cases it is important to note that there is progressive rise of the pulse-rate *at once*, that there is no rise of temperature at first (though there may be later if the patient survives), and that there is no large tumour and no marked fixation of the uterus as there is in inflammatory cases.

Occasionally appendicitis and ruptured gastric ulcer will have to be distinguished from hæmorrhage cases. The latter particularly may give rise to great difficulty, and we can only insist that the usual history of these cases and the general trend of the symptoms must be carefully scrutinised.

In appendicitis the localisation of the greatest pain to the right side, the possible presence of a lump there, the rise of temperature, and absence of progressive pallor should usually serve to distinguish the lesion.

In gastric ulcer cases the history of dyspepsia and vomiting, possibly hæmatemesis, the onset of acute symptoms *after a meal*, the abolition of the liver dulness, and the greatest pain in the epigastrium may help us to differentiate such cases.

*Axial rotation* of an ovarian cyst is said to occur in about 10 per cent. of all cystic ovarian tumours. It gives rise to such important and serious results that early diagnosis may have a great bearing on the treatment and prognosis of the case. The acuteness of symptoms depends very much on the tightness of the twist in the pedicle and the rapidity with which it has occurred. As the first effect of tight twisting is interference with the venous return from the tumour, hæmorrhage into the cyst occurs from the great congestion produced, and the cyst may even burst, giving rise to free peritoneal bleeding. This, however, is not a common result; as a general rule the bleeding is confined in the walls and cavities of the tumour. This, of course, gives rise to great tension and acute pain in the abdomen. It is very quickly followed by peritonitis all over the surface of the tumour, producing universal adhesions, and the tumour itself may become infected, organisms finding an entrance from the adherent bowels. The severity of the circulatory changes may give rise to partial gangrene if the case is undiagnosed with correspondingly important symptoms. We are, however, more particularly concerned with the diagnosis of the case at its onset or soon after. We first notice that there is sudden pain, some collapse, which tends to pass off in most cases, but progressive if free peritoneal bleeding occurs, followed by rise of temperature and signs of peritonitis. We also note that these symptoms are associated with an obvious tumour. Practically this is the only important acute pelvic lesion which is associated with an obvious tumour. The tumour fluctuates, generally has a thrill, and shows the areas of resonance and dulness commonly associated with ovarian cysts. It must not be forgotten, however, that free fluid may rapidly form in the peritoneum and may thus obscure the usual signs and even the tumour itself. If the patient is known to have an ovarian cyst these signs, of course, are conclusive, but it often happens that the first knowledge of an ovarian cyst is discovered when axial rotation has occurred. Bearing in mind that axial rotation is a common accident in relation to pregnancy or labour, or may be associated with some sudden movement, it must be admitted that this lesion is perhaps the easiest to diagnose of all the acute abdominal conditions of pelvic origin.

*Rupture of an ovarian cyst* is a comparatively rare accident, and, like axial rotation, may be caused by pregnancy or labour, though direct violence and sudden movements are responsible for more such accidents than in cases of axial rotation. The symptoms produced will depend very much on the kind of tumour and the amount of bleeding produced. Unilocular cysts with

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watery contents may cause very little disturbance after the first acute pain and sensation of something giving way. The fluid may be absorbed and excreted by the kidneys, and the tumour may slowly refill. On the other hand, the pseudo-mucinous contents of a multilocular cyst or the peculiar "brew" of a dermoid may cause peritoneal irritation and even mild peritonitis. If only one loculus of a multilocular cyst is burst leakage may go on for a time, or the hole may be plugged with one of the daughter cysts. The diagnosis may be difficult or impossible, unless the patient was known to have a cyst and it can be shown to have disappeared. On the other hand, if a large vessel is ruptured in the accident serious peritoneal hæmorrhage will occur with its characteristic signs. If the patient was not known to have a cyst the case may then be mistaken for ruptured ectopic gestation or ruptured gastric ulcer, and no doubt there will be great difficulty in making a diagnosis. If, however, the signs of intra-peritoneal hæmorrhage are clear, the diagnosis matters very little, for the abdomen must be opened to stop the bleeding. The secondary effects of rupture of an ovarian cyst do not concern us here, but it must be remembered that peritonitis may ensue, and that malignant growths may be disseminated from papillomatous cysts.

*Rupture of a pyosalpinx* is a very rare accident for the reason that most cases of this nature have very thick walls the result of interstitial inflammation, and also that as the pus collection enlarges the inflammatory process adds to the thickness of the wall by organisation of the peritoneal adhesions around. On the other hand, it is not uncommon for ulcerative processes to take place in the wall of a pyosalpinx and gradually to produce such thinning that a small leakage of pus occurs. This is a very chronic process, and may possibly account for some of the cases of recurrent pelvic peritonitis in connection with the pelvic organs. This is not quite what one pictures as a rupture of a pyosalpinx, One pictures this accident as the bursting of a somewhat thin-walled pus sac, flooding the pelvis with inflammatory products and setting up an intense peritonitis which is more likely to be general than local. Such an accident must be excessively rare, and would necessarily be extremely difficult to diagnose. We can only make a provisional diagnosis in such a case after a careful survey of the history and physical signs. We should expect a history of previous pelvic inflammation, possibly of long-continued vaginal discharge, and an absence of any question of pregnancy. We should expect signs of peritonitis with its quick pulse, rise of temperature, vomiting, and great rigidity of the abdominal muscles. It would be most likely impossible to distinguish it from a ruptured gastric ulcer or a perforating appendix.

*Local peritonitis* is, perhaps, the commonest of all the acute abdominal conditions the gynæcologist has to deal with, but it is not often of so great importance as rupture of a gestation sac, because it is rarely generalised, and, therefore, not often leads to a fatal result. Apart from the cases of pelvic peritonitis caused by lymphatic infection from the uterine cavity, it is almost always the result of a spread of infection from the uterus *viâ* the Fallopian tubes to the peritoneum. For this reason it is either gonorrhœal or due to other organisms which have caused a septic endometritis. An important point to note is that salpingitis gives no very obvious symptoms until the peritoneum becomes infected, and then the case fulminates as one of pelvic peritonitis. Whatever the organism which sets up the lesion the symptoms are fairly well defined, and are not likely *at the onset* to be taken for those of a ruptured gestation sac. There may be an initial rigor with great pelvic pain and rigidity of the lower part of the abdominal muscles. Some degree of vomiting always occurs at first, and the temperature is high, the pulse also being raised in frequency. On examination we shall find a pelvic swelling of indefinite outlines, sometimes rising above the pelvic brim, and due to a fluid exudate, which afterwards coagulates to form a thin, jelly-like material. This naturally fixes the uterus in a central position, and gives the well-known sensation as if that organ was set in plaster-of-Paris. If the tumour rises above the pelvic brim it is always more or less resonant on percussion, because of the intestines adherent to it. If, however, the infection is an excessively virulent one it may occasionally happen that general peritonitis is set up, in which case the symptoms are much more serious. The important guides to diagnosis in such a case will be complete fixity of the abdominal muscles with tympanitic resonance all over, constant vomiting, and the thready pulse, which progressively rises in frequency. The

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diagnosis between these two possibilities is important because the latter requires immediate operative treatment, the former does not. The method of infection in some of these cases is not always clear. They occur sometimes in young girls during menstruation, and these are the cases which have often been stated in text-books to be due to "cold during menstruation." We do not actually believe this now, but we can realise that there is some underlying truth in the statement. Menstruation alone lowers the resisting powers of the individual, and a chill at this time may be expected to still further reduce these powers. Therefore, it is not unreasonable to believe that there may be a greater likelihood of infection occurring during menstruation. Whether the infection really occurs as the result of a salpingitis, or what is more probably the case as a result of bacterial migration from the intestine, at present is not known. It is a fact, however, that a certain number of cases of suppurative peritonitis do occur during menstruation in which no infective lesion can be found in the appendix or the pelvis.

*Treatment.*—From what has been already said it will be clear that there is not much room for argument about treatment in these cases when a diagnosis can be made with any degree of certainty. However, as diagnosis is often difficult in the extreme the question of treatment often has to be faced with great anxiety, and a big responsibility rests with the surgeon, upon whose decision the treatment rests.

Acute and progressive intra-peritoneal hæmorrhage, whether it is the result of ruptured ectopic gestation or ruptured vessel in the broad ligament, or tumour, requires very prompt treatment if life is to be saved. There is very little hope that the hæmorrhage will cease as the patient becomes exhausted, for this is Nature's only way of arresting bleeding in such a case. When once progressive bleeding has been diagnosed the sooner the abdomen is opened and the bleeding vessel secured the better it is for the patient. The chance of doing this with success can only be early in the case, before the patient has lost a fatal amount of blood. It is of great importance that the patient should not be moved more than can be helped, and especially should removal to a hospital be avoided if possible. Sometimes in these cases bleeding is temporarily arrested by coagulation, but is at once re-started if there is much disturbance of the patient. It cannot be affirmed that no case ever recovers without operation when progressive bleeding has been diagnosed, because there are many cases on record where the patient has seemed to have passed the boundary when an operation could be done successfully, and yet has recovered. Clearly to operate on a patient whose pulse could hardly be felt and who was unconscious would be only to court disaster. Such cases can only be treated by bandaging the limbs, infusion of saline solution, and with opium if they show any signs of recovery. There will always be a danger of re-starting the bleeding in such a case, and absolute immobility is the only way to avoid it. On the other hand, if the patient is clearly getting worse from one ten minutes to the next, but yet is not severely collapsed, to open the abdomen rapidly and secure the bleeding point is a true surgical principle and one that has been attended by brilliant results. It need hardly be said that fluid must be got into the patient after this operation, and it may be done very simply by leaving saline solution in the abdominal cavity when sewing up. If fluid must be got into the patient's vessels rapidly on account of signs of a failing heart, then it must be infused directly into a vein and the heart stimulated by strychnine. Bandaging the limbs and elevating them with the pelvis or "auto-transfusion" will be a good means of keeping the blood as much as possible to the brain, and a fair amount of pressure may be put upon the abdomen to prevent the blood accumulating in the portal area. Whereas there can hardly be two opinions as to the correct treatment where bleeding is progressive, we cannot say the same in ruptured gestation sacs where the bleeding appears, from the appearance of the patient, to have ceased. This can only be decided by observing the patient at short intervals after the initial acute attack. The pulse will remain stationary or will slowly drop in frequency if bleeding has ceased. According to Herbert Spencer and others it is hardly ever necessary to operate upon cases of this kind. These authorities say that with absolute rest procured by opium, raising the foot of the bed, auto-transfusion, etc., these cases will always recover. The blood effusion into the general peritoneal cavity rapidly absorbs and only a proportion will remain in the pelvis as an encapsuled hæmatocele. These statements must

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not be taken too literally, for it is by no means uncommon for a second hæmorrhage to occur with a fatal result. Particularly may this be the case when the ovum has not entirely lost its attachments to the tube and does not perish but goes on developing. From another point of view the expectant treatment is unsatisfactory, and that is the length of time such patients take to recover. An encapsuled hæmatocele undoubtedly will be absorbed, and certainly does not often give rise to serious trouble, such as suppuration. It must not be forgotten, however, that these patients are not free from pain in the pelvis; they are invalids for several months in many instances, and therefore if in poor circumstances are quite unable to earn their living. For these reasons it is often more satisfactory to operate than to leave the case alone. If the patient's condition is improving after the initial attack, a rapid laparotomy will not seriously endanger her life, and provided the operation is an aseptic one there is practically no risk apart from that of shock. The time to operate in such patients can only be decided by a careful survey of every individual case.

The cases upon which it is agreed on all hands operations should be performed are those in which ectopic gestation is diagnosed before any serious hæmorrhage has occurred. The initial pelvic pain due to small hæmorrhage into the ovum itself often is bad enough to warrant medical advice, and if a diagnosis can be made at this time a laparotomy is attended by very little risk indeed, in fact no more than in an uncomplicated ovariotomy. No doubt it is true that some ectopic gestations never give more trouble than this, that they are converted into moles, no further bleeding occurs, and the mole is slowly absorbed. This is a view I have held for some time past, but as it is incapable of proof without unnecessary operations I have no real evidence to offer in support of it. Nevertheless it is impossible to say which tubal moles will give rise to serious bleeding and which will not, and therefore it is better to operate if such a condition is diagnosed.

The operation presents no difficulties; all that has to be done is to open the abdomen, tie the ovarian artery and remove the gravid tube, sewing over the peritoneum afterwards.

The same treatment holds good for progressive bleeding from a ruptured vein in the broad ligament; it must be tied, and if there are many varicose veins near they should be excised. In bleeding from large vessels or tumours—fortunately very rare accidents—considerable discretion is necessary when operating. If the patient is badly collapsed from hæmorrhage all that should be done is to secure the bleeding vessel, as any prolonged operation for the complete removal of the tumour will place the patient in great danger. No patient who has lost much blood can stand a long operation.

*Torsion of an ovarian pedicle* with hæmorrhage into the cyst and peritonitis on the surface requires operation as soon as diagnosed. This is necessary because the tumour may become gangrenous, the peritonitis may spread and become general, the tumour may suppurate, and the adhesions will become more and more firm the longer the case is left if no worse conditions supervene. These operations present no particular difficulty and the results are very good if no complications have occurred. It is wise in transfixing the pedicle to get beyond the large thrombosed veins generally found in it if possible. The possibility of tying the ovarian veins higher up in the abdomen should be considered, so as to be sure of getting beyond thrombi. This is important, because there is a distinctly increased risk of pulmonary embolism occurring in these cases, and this is not diminished by tying a vein through the middle of a large thrombus.

The adhesions in these cases are often universal. but are easily broken down as a rule and do not require any ligatures. No drainage is necessary in such cases however adherent the tumour, the only exception being the formation of pus outside the tumour—a condition which very rarely occurs.

*Rupture of ovarian cysts* if diagnosed should be operated upon, because it is impossible to foretell the character of the fluid effused or the character of the cyst, whether benign or malignant. Progressive bleeding in such cases will show itself by symptoms, and, of course, will be an imperative reason for immediate operation. Leaking cysts can rarely be diagnosed, but as there will be an obvious tumour in such cases, the symptoms will usually make a laparotomy advisable, and the condition will then be revealed.

The treatment of peritonitis at its acute onset depends chiefly upon its local or general characters.

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Briefly it may be said that acute pelvic peritonitis rarely requires immediate operation, whilst general peritonitis is almost, if not quite, invariably fatal if left alone. Local peritonitis of gonorrhœal origin particularly does not require immediate operation, though it is fair to say that there is not much risk in the operation itself. The acute attack of pelvic peritonitis, whatever its cause, very soon becomes localised as a rule, and the patient begins to improve. There are, however, cases in which it is impossible to say at first whether localisation has occurred or not, and in them an exploratory laparotomy is often necessary. If no general peritonitis is found on doing this and the pelvic exudate is shut off by adhesions it is wise not to interfere with them from above. The reason for this is that the difficulty of the case depends upon unusually severe symptoms such as high fever, continuous vomiting, and signs of a more generalised septicæmia. To attempt to break down adhesions from above would almost certainly produce a diffuse peritoneal infection. It is better in such a severe case to close the abdominal incision and then make an opening into the pelvis through the posterior vaginal fornix. In this way the pelvic exudate can be drained and as a rule very satisfactory results occur. Such an operation does not make a future salpingo-oöphorectomy any more difficult, and undoubtedly shortens the patient's illness and may produce an absolute cure.

In cases where no operation is proposed all the treatment necessary is to relieve pain, to apply heat and to keep the bowels acting. Morphia must be given for the pain, but the least quantity which will suffice should be given as it has a distinctly deleterious effect in constipating and masking symptoms. Heat may be applied by vaginal douches, at least two quarts at a time, and by hot abdominal compresses. Saline purgatives are the best to keep the bowels acting.

If general peritonitis is present nothing short of abdominal section will give the patient a hope of recovery. If the source of the infection is the Fallopian tubes they should be removed, in fact any source of infection should be removed, if possible, bearing in mind that the operation must be rapid. All pus and fluid should be mopped out quickly with swabs, but no attempt should be made to wash the peritoneum clear. This will only have the effect of washing infected material into parts of the cavity which, perhaps, have hitherto escaped. Drainage must then be provided for, and, remembering the points where fluid exudates collect, we drain the pelvis through the vagina and the lumbar recesses through the loins, whilst the central laparotomy wound may also be used to provide general drainage if necessary. After this operation two great essentials must be carried out, namely, the Fowler position, that is, keeping the patient propped up in a sitting posture as soon as the effect of the anæsthetic has gone, and some form of continuous saline infusion. The Fowler position ensures the gravitation of all fluids to the lower abdomen and pelvis, and is the best known means of preventing infection of the diaphragmatic peritoneum. Continuous saline infusion ensures a plentiful lymph flow, promotes diuresis, and thus helps absorption from the peritoneum and rapid elimination of toxic products. Nothing can be worse for general peritonitis than a sluggish circulation of blood and lymph with accumulation of toxines. Purgatives will, undoubtedly, be indicated where there is no diarrhœa, for they prevent paralytic distension of the intestines and promote the peritoneal lymph flow. Saline infusion may be given continuously by cannulæ inserted in the legs attached to an irrigating apparatus raised only about a foot. All that is necessary is to keep a slow continuous flow of saline solution at about the rate that the vessels can absorb it. In this way five to seven pints can be got into the system in a day. Continuous infusion *per rectum* in the same manner is recommended and carried out by some operators. If continuous infusion cannot be used on account of intolerance, saline enemata should be slowly injected every four hours until improvement begins.

In conclusion, I fear that my remarks are somewhat disjointed and no doubt incomplete in many details, but it is my hope that the essential principles have been dealt with, and that the manner of their enunciation has not bored you.

*July 20th*, 1908.

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# A CASE OF ENCHONDROMA IN THE TENDON-SHEATH OF THE FINGER.

By R. R. WALKER, B.C., M.A.,
Late House Surgeon, Guy's Hospital.

A CASE presenting some unusual points was admitted under Mr. Arbuthnot Lane at Guy's. The patient was æt. 54 years, a gardener by trade.

At the age of fifteen he had what he described as the "rheumatics" in the second finger of the right hand, and the finger became somewhat swollen at this time; there was no history of injury.

The finger was tender, and there appeared a small lump on the flexor aspect of the proximal phalanx of the finger.

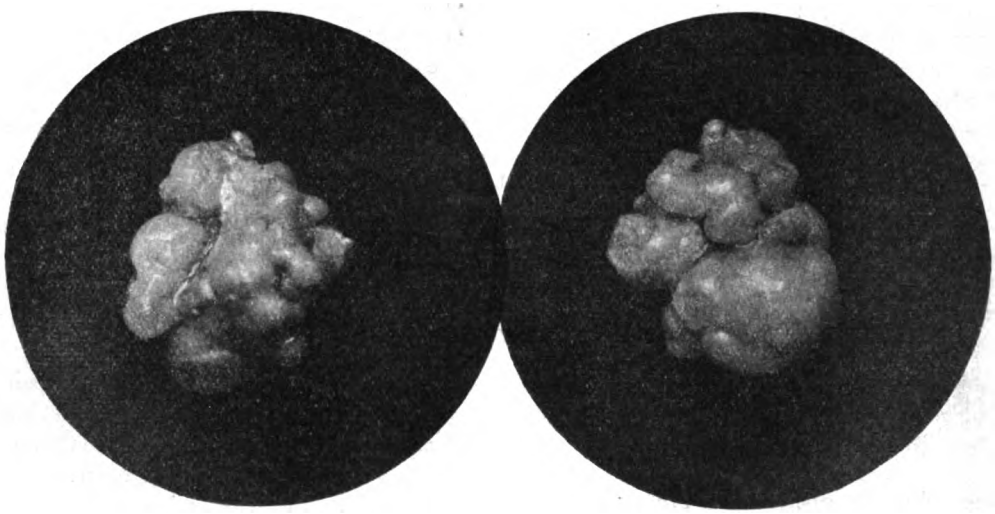
For many years this gave him no pain, and only caused some inconvenience by preventing complete flexion of this finger by its size and position. On February 28th of this year the patient injured the swelling with a hammer, and since this time it has been painful and tender.

On examination there was found to be a large swelling over the palmar surfaces of the two proximal phalanges of the second finger of the right hand. The skin over the surface of the swelling was thickened, but not adherent to the tumour. There was no fluctuation, and the tumour could be moved from side to side on the phalanges, but little, if at all, in the long axis of the finger.

The movements of the finger were limited mechanically by the size of the swelling.

An incision was made in the middle line of the palmar surface of the finger in the line of axis of the finger, and the tendon-sheath of the flexor tendons exposed; this was opened, and there was found to be a pearly white tumour, of the same size as the photograph shown here, of a very irregular mulberry-like shape.

The flexor tendon ran in a groove of this tumour, between the tumour and the bone. The groove for the tendon shows plainly in photograph A.



A. B.

A. Dorsal surface showing groove for flexor tendon. B. Palmar surface.

The whole tumour lay in the sheath, and articulated with the head of the proximal phalanx, but was adherent nowhere, either to bone or cartilage.

The tumour was removed easily, and on being lifted out of its bed the flexor tendon lay exposed.

The patient made an uneventful recovery, and has good use in his finger.

The tumour, on microscopical examination, proved to be an enchondroma.

I am indebted to Mr. Lane for permission to publish this case.

*July 20th*, 1908.

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# THE CLINICAL JOURNAL,

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** Specially reported for the Clinical Journal. Revised by the Author.*

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## TWO CLINICAL LECTURES ON INJURIES TO THE HEAD.

Delivered at St. Bartholomew's Hospital.

By ANTHONY A. BOWLBY, C.M.G., F.R.C.S., Surgeon to the Hospital.

### LECTURE I.

GENTLEMEN,—I want to speak to you for a few minutes to-day about a case of injury to the head, which was admitted to my wards a fortnight ago. The story, briefly, is this. A girl, æt. 13 years, fell on her head from a height on to the floor. The floor appears to have been a concrete one: whether there was anything upon it where she fell the history does not state. She had a scalp wound, and apparently for the time being she was "knocked silly." Whether she saw a doctor then or immediately afterwards I do not know, though I believe she did not. But the wound on her head was bandaged, and for a period of, at any rate, ten days she did not seem to be materially ill. No particular precautions seem to have been observed to keep her in bed or treat her as an invalid. Soon after ten days had elapsed, however, she began to get evidently worse, she had more headache, her temperature rose and she had a curious condition of semi-stupidity, so that it was not at all easy to converse with her, for she seemed unable to give ready answers, Soon after that the doctor who saw her examined her head, and at once sent her into the hospital with a note stating that she had a depressed fracture of her skull. In the evening of that day I saw her and heard the story. On speaking to the girl I found she was very heavy and lethargic, not at all inclined to respond to anything that we said to her and unwilling to utter a single word in reply. That was a condition which indicated some alteration in her brain, and realising that this lethargy suggested something more than could be attended

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to without an anæsthetic, we took her into the theatre, shaved a portion of the scalp, and having rendered the parts as aseptic as we could, we examined what was now a suppurating wound. On putting one's finger into it one encountered fractured and depressed bone, and in the front of this wound there was protruding some softened and suppurating brain. The situation of the wound was in the left frontal region, rather towards the vertex. This wound was enlarged sufficiently to ascertain the position of the fractured fragments, and it was found then that one portion of the bone of the skull was depressed over an area of two or three inches below the level of the adjacent bone. Anteriorly the fracture ran forward, and a piece of bone appeared to be entirely missing, so that one could put one's finger through a hole in the skull of the size, perhaps, of the end of one's index finger. This was the condition, then: This patient evidently had been going about with a compound comminuted fracture of her skull, with laceration of the dura mater, and with the brain torn and injured. When we came to further enlarge the wound and apply a trephine and cut out a portion of bone so as to enable us to introduce an elevator and lift up the depressed piece of skull, we found that the missing piece of bone was deeply buried in the left frontal region, and it was only with a good deal of difficulty that we extracted it from deep in the brain. It was necrosed, because it had been separated for a fortnight, and it necessarily was a source of suppuration in the brain.

The first thing that this case teaches you is, that in every wound of the head or scalp where there is the least reason to suppose from the history of the accident that there may be any injury of the skull, you must examine with your finger the injured part at the bottom of the wound. No probe can tell you as much as your finger can. Understand, I do not mean that in every case you are to enlarge a wound sufficiently to admit your finger—I mean that in every case in which the wound is large enough, your finger is infinitely a better instrument than anything that can be devised in the way of a mechanical tool. The finger-tip has so much greater sensation in it than anything else you can introduce, that there is no doubt it is very much better than a probe. Now, you may think it is an extraordinary thing that a patient should be able to go about with a fractured skull, but you must remember that people can go about with fractured skulls, and even with comminuted fractures of the skull, as in this particular case, and that an injury of the skull alone does not necessarily carry with it any particular or serious symptoms. Some of you may know that Percival Pott was surgeon to this hospital a great many years ago, and this is his book which I have in front of me. More than a hundred years ago he quite recognised the fact that people can go about with comminuted fractures of the skull, and he very clearly points out that where there is an injury to the head which does not injure the brain or dura mater, the mere fracture of the skull may give rise to no very particular symptoms at all, and he relates the case of a boy who fell from a cart loaded high with hay and pitched on his head. "The blow stunned him for a few minutes, and then he got up again and walked home with the cart. As he made no complaint, his master took no notice of this fall and the boy followed his daily labour in the farm-yard. At the end of a fortnight he came to see the surgeon with a swelling on the upper part of the head. The tumour appeared to be full of matter, and the surgeon divided the scalp and let out a considerable quantity and passed his finger in to examine whether the cranium was bare or not. He found it not only bare but considerably comminuted. Distinct pieces of bone were so loose that they could be taken away without resistance. So much were they as altogether to make nearly one third part of the parietal bone. The dura mater was clean, the boy had no symptoms from first to last. He came to the surgeon's house every day to be dressed and was also in the farm-yard daily." Now I do not suggest that it is a good thing for a boy with a fractured skull to go to the farm-yard daily, but I suggest that in cases of this kind, where there is no injury to dura mater or brain, a patient with fracture of the skull may easily walk about. You must keep clearly before your minds, therefore, that a mere break of the skull need not necessarily carry with it any particular symptoms or any special disability.

Fissured fractures of the skull are the simplest fractures of all, and there can be no doubt whatever that in many cases skulls are fissured without there being any external sign of it. You may say,

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"How do you know, or why do you suppose that people suffer from fissured fractures of the skull without a surgeon being the wiser for it?" You can tell that it is so easily enough, because there are plenty of cases where a patient is admitted to hospital with some injury which after a time results in his death, apart from any injury to his head, and only on making the post-mortem examination is a fissure discovered. I am telling you my personal experience, for I have seen several cases where no suspicion of any fracture has existed, and when the patient was in the post-mortem room the fissure was discovered accidentally. It is then only begging the question to say, "What are you going to do in the treatment of fissured fracture of the skull when there is no external wound?" The answer to that is, that if it is not to be felt or seen it is not treated at all. A man who has had an injury to his head and has had extravasation may or may not have a fissure of the skull, and if he has a fissure and nothing else, it does not matter so long as it is not complicated by a laceration of the brain or a tearing of blood-vessels, for the treatment for the simple fissure is nothing at all. In other cases there is a fissured fracture of the skull with an external wound, and you have in that case to deal with a compound fracture. And considering that on the other side of the fissure is the dura mater and beyond that are the coverings of the brain itself, it is evident that in this part of the body more than any other, you have to be specially careful that your wound is made as aseptic as possible and treated most carefully.

Now, I have spoken to you of fissured fractures of the skull as of no importance except as regards sepsis. But I want to allude to a condition which is important in infants. This is the skull of an infant, and only six weeks before this child's death it had a fissure of the skull. You will see that in the course of a few weeks that fissure has become enlarged, so as to measure about one and a half inches in length and nearly an inch in breadth. This fissure in fact has gradually, in the case of the infant, become converted into a large hole. Here is a picture of the head of another child, showing the result of one of these fractures and the swelling caused by the protrusion during life. This is, therefore, a curious complication which may ensue in fissured fracture of the skull in infants. A fissure in the infant's skull may soon become stretched by the protrusion through it of fluid from either ventricle or subarachnoid space, and by the constant pulsation the soft bone is rapidly absorbed, and in the course of a few days there may be noticed a pulsating swelling, and if no measures are taken to prevent extension of this, this pulsating swelling will increase as the fissure becomes larger, and ultimately there will be a large fluid swelling on the side of the child's head—a "traumatic cephal-hydrocele." I want you to remember this because when you have to do with a baby who has had an injury to the head and an extravasation of blood, and it has a hæmatoma, you must keep watch to see that in the course of a week or two that hæmatoma subsides. If it does not subside, be on the look-out for it beginning to pulsate. The pulsation means that there is a communication between the swelling outside the head and the inside of the skull. I have seen these cases at this stage, and I can tell you, that if the child's head is firmly bandaged and a sufficiently elastic form of pressure be applied with cotton-wool, the whole of the symptoms will subside, and this very serious complication will not result. But it is too little appreciated that whereas fissured fractures in adults, apart from sepsis, give rise to no material symptoms or trouble at all, in infants there is this associated trouble in a few rare cases.

But if we leave fissured fractures and go a step further and consider what to do when the bone is depressed or broken, what then? The more the bone is depressed the more necessary it is for you to elevate it into its proper place, in other words to "set the fracture," for after all that is what you are doing. If a man has a broken tibia, or femur, or humerus, and the ends overlap, you of course do your best to put them into position. And the only way you can do this in the case of a fracture of the skull with displacement of fragments of bone is to use an elevator, and if necessary you may have to cut out a portion of the skull bone to enable you to introduce an elevator, and you may use a trephine for the purpose. If the bone is comminuted there again there is nothing different in the treatment of fracture of the skull compared with that of fracture of any other bone in the body. If you are brought to a case of fracture of the tibia which is comminuted, with pieces of bone torn away, and there is a piece of bone absolutely loose in the wound, you take it out. If that is the

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condition of affairs in the skull you take the piece of bone out from there also. But in the girl whose case I have mentioned there was not only a comminuted fracture of the skull with an external wound making the fracture compound, but there was also a tear of her dura mater. That takes us a step further. There is, therefore, here an opening up of one of the large serous cavities. This wound is analogous to a wound of the chest or of the abdomen which opens into the pleura in the one case or into the peritoneum in the other. It has opened up a serous cavity in which suppuration, if it occurs, and in which sepsis, if it exists, is liable to be diffused. Here you have, therefore, a complication; you have a perforated wound of the skull, a wound which has gone through the skull and dura mater, and which has opened up the parts beneath. All the more need, therefore, to take care that every possible precaution is observed against sepsis, and that every piece of bone is taken away with the utmost gentleness.

I want to remind you of what was done at this hospital very many years ago, and I will read you an extract from what Pott wrote which is as true to-day as it was when he wrote it. He says: "The brain is wounded, either by the instrument or the body whereby the skull is broken, or by the broken parts of the cranium. But let the wound or fracture be what it may or whatever other circumstances may happen to attend, the chirurgic treatment is short and plain, namely to remove all such parts of the broken skull as may compress, wound or irritate the brain or its membranes, to take away all such extraneous bodies such as can easily and without violence be got at and extracted, and to make such an opening as may most conveniently serve the purpose of discharging blood, serum or matter. When all these things have been done and the patient has been put under proper treatment, both as to diet and regimen, the surgeon has done his duty, and can say with Pope:

'Thus far was right,
The rest we leave to Heaven.'"

His treatment was exactly the treatment which you should carry out at the present moment. It is true that he did not know anything about antiseptics or anything about bacilli, but his actual work was correct. He tells you the necessity for care in examining the wound, and taking away foreign bodies and making a sufficiently large hole in the skull. He says with regard to dressings that they should be soft and light and not consist of anything greasy, and not be applied in such a manner or quantity as to obstruct the free discharge of fluid, and you will find that he threw aside all the quack remedies which have always existed for the treatment of injuries of the head, or indeed of any other part of the body. And following his advice, I would again remind you that the important thing in a case of injury to the head is to examine the wound most carefully and thoroughly so as to ascertain the extent and nature of the injury, for unless you ascertain that, you may easily be deceived by the apparent well-being of the patient, who may not show at first any evidence to indicate there is anything besides the external wound. Yet there may be, as in this girl, an injury to the bone, and tear of the dura mater and a lacerated brain, though none of these things may be discovered unless you examine the wound to ascertain the condition of affairs. And do not forget that if the wound is large enough there is nothing so good as your finger for this purpose.

Now let me say a few words more. Injuries of the skull may be complicated as we have seen by direct injury of the membranes of the brain by the broken bone itself. And the part injured may be a blood-vessel, and it may bleed. A broken piece of bone may tear a meningeal artery, and that artery may consequently bleed, and the blood may strip the dura mater and may collect between the dura mater and the bone, and its quantity continually increasing it may press upon the subjacent brain. So the brain may be compressed by a clot between the dura mater and the skull, the result of some broken bone tearing a vessel which bleeds.

We thus come to the condition which is talked of as "compression of the brain." There are two terms which you must keep clearly in your minds in speaking of injury to the head: concussion and compression. Let us speak first of all of concussion.

"Concussion of the brain" is a purely clinical term, but I will tell you a definite pathological condition which is most commonly associated with it. Concussion of the brain is much better spoken of as "contusion" or bruising of the brain. When

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the brain is injured the term "concussion" suggests that the brain is simply shaken. But that is not enough. To cause definite symptoms there is practically always a definite lesion. When there is the clinical condition which we know as concussion of the brain the least which you will see, or would see if you were to make a post-mortem examination, is such as is represented in the picture I show you. The under-surface of this brain is contused and the temporo-sphenoidal lobe is discoloured and bruised, and there is shown a very slight escape of blood underneath the pia mater. That is exactly analogous to what you see when a person has contusion of any part of the cutaneous surface of the body. When a man, then, has "concussion" of the brain you must interpret that to mean that if you could look inside his head you would see that his brain was contused. A contusion is a "lacerated wound." You will find that definition in most text-books of surgery. Now the main symptom of "concussion" is shock. What are the symptoms of serious injury to any part of the body, to abdominal viscera, thoracic viscera, or the cranial viscus? Shock. What do you mean by shock? You mean that the patient is in a condition of greatly depressed vitality. He probably is lying down and is not able to sit up; he probably feels cold, his temperature is actually below normal, and his skin is often cold and clammy. His pulse is usually small and it may be fast or it may be slow or irregular, but it is small always. These conditions only indicate that the patient has suffered from shock. What is to tell you that the injury is an injury to the brain, and that the shock is due to injury of the brain? What is to guide you in saying that he has "concussion"? The main thing is that such a patient has impaired cerebration; he does not think quickly or appreciate what is said to him quickly. He is dazed. He may be conscious, but he may take a long time to appreciate the question which is put to him, and you may have to be very persistent before you can penetrate the dulness of his mind and extract an answer. That means that at first he cannot realise what it is you are asking him, but by persistence you may get him to realise it, but even then he may not be able to tell you where he lives, or the number of his house. That would be only a slight case. But this impaired cerebration may be so marked that he may be unconscious, and can only be roused with difficulty, so that he is what is called "insensible." But you may be unable to rouse him at all. Yet even though you cannot rouse him he may be able to move his arms or his legs. If you were to pinch or to prick him or stimulate him in any other way he would move even though he were insensible. And this condition of insensibility may last for a variable time. In a slight case it may last for only a few seconds. In a severe case the man may be unconscious for weeks, and every gradation between those two extremes may be found. And nothing is more important than to realise that in cases of severe "concussion of the brain" there is practically always a certain period of loss of memory, so that a man will often be unable to give you a description of the actual accident at the moment that it happened. Here is a typical case of very slight loss of memory following upon concussion. A man was admitted into the hospital suffering from concussion of his brain. He soon became sufficiently conscious to answer questions, and after he had been put to bed and made comfortable he volunteered the information that he had been going up the ladder of a scaffolding when he slipped from the ladder and fell down. When we came to inquire from others who had seen the accident, it turned out that our patient had gone up the ladder, that he had got on to the staging where he was assisting in building a house, that he had deposited there what he had carried up, and then stepped on to a loose plank, and that plank had given way and precipitated him to the bottom of the building. He knew nothing of this, for the last thing he remembered was going up the ladder; he did not remember reaching the top of it, nor what he did when he got there. Evidently he had lost recollection of about one minute. In another case a man was admitted here, having been thrown off a van which he was driving along Holborn Viaduct. He was insensible. He remained insensible, or, at all events, he could only be slightly roused, until the next morning. But when we spoke to him he explained that the accident had happened on the south side of London, near the Elephant and Castle, and he had no recollection of anything after that. Yet it turned out that this man had driven his van from the Elephant and Castle, across south London and London Bridge, and through the City, but he did not remember having done so.

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He had lost memory of that half hour or so which must have elapsed since he left the Elephant and Castle. So remember that in a case of concussion, the thing which points to the brain having been injured is the presence of shock and slow cerebration and inability to remember what happened immediately before the accident. In many cases of severe brain injury the loss of memory may be so severe as to extend to events happening for days, or in curious cases, to weeks or months. But when the patient is coming round and begins to move about he complains of headache and giddiness, and if he sits up in bed his head swims; he hates a bright light and does not like a noise. That indicates hypersensitiveness of his brain, and the more his brain has been injured the more these things will affect him. Suppose for a moment that you said to such a patient, "You may get up and walk about," what harm would come to him? One point is that by allowing him to walk about you would prevent the brain having that rest which it ought, and the more you prevented the injured part having rest the more likely would it be to bleed. There is another thing. If a man has an attack of apoplexy, or hæmorrhage into his brain from a diseased blood-vessel giving way, if he survives, he begins to have fever, and if you were to examine his brain at this stage you would see that hyperæmia had set in, with increased vascularity, and soon after that there is a further softening of the brain from inflammatory processes. And many a man dies after apoplexy from softening of the brain, which sets in some two or three days after the original hæmorrhage. And if you were to make a post-mortem examination of him you would find a good deal of disintegration of the surrounding brain. He has died, not directly from the original hæmorrhage, but from the disintegration of the brain which has followed the irritation produced by blood-clot and hyperæmia of the surrounding cerebral tissue. And so in concussion, if you do not keep the man quiet, if you let him be up and move about, he may die. Therefore, in all these cases you have to keep the damaged part at rest, keeping the man at the same time on sufficiently low diet. There is one other precaution which you must always take, and that is, give him something which will freely open his bowels. There is no doubt whatever that in proportion as a man has had injury to the brain it is very difficult to get his bowels to act. If you look at the prescription sheet in a large number of cases of head injury, you will find that dose after dose of aperient medicine has been ordered. The best thing to begin with when the man is only semi-conscious is a dose of calomel. But a dose of calomel which would be sufficient in health as a purgative is absolutely insufficient in a case of concussion of the brain. If you give a healthy man two grains of calomel it will be enough to cause the bowels to act freely. But if you give a man with a severe injury of the head six or eight grains of calomel you may find that it will not act. You may give two ounces of senna draught or an ounce of castor oil without effect. Remember, then, that these people with injury of the brain are very difficult to act upon by means of aperient drugs. I cannot tell you exactly why it is, and I do not much care. The important thing is that you must give these people large doses in order to obtain any good result. This difficulty is notorious in such cases, and until the bowels have freely acted there is always a period before which the symptoms, and especially the headache, do not begin to improve. Beyond ordinary precautions, after that, there is very little to be done for these patients.

I want to tell you now that in proportion as a man has had an injury to his brain which is serious, and the symptoms of which have been prolonged, he has to be excessively careful afterwards, perhaps for months, perhaps years, or it may be during the whole of his life, in certain important particulars. What particulars are they? Precautions in regard to alcohol first, and in regard to exposure to excessive heat secondly. If there is one thing which is certain, it is that in a person who has had a severe brain injury, a very small quantity of alcohol will make him as excited, or as drunk, or irresponsible for his actions as a very large quantity would have previously made him when he was in robust health. There is no doubt that almost all people who have had serious brain injuries have to be extremely careful about taking alcohol. And it is best to advise them to completely abstain from alcohol in any form. A man also who has had sunstroke sufficiently severely is liable, on the slightest imbibition of alcohol, to become practically irresponsible for his actions. There is apparently, for some

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reason or another, either in cases of sunstroke or in severe brain injury, such an alteration in the whole vaso-motor system of the brain that the slightest thing puts the patient off his balance. And the next thing is that you must remember that the patient must be careful about exposure to heat. There is no doubt that people who have had injury to the brain are not able to stand so much exposure to the sun as other people. Then these patients with severe brain injuries may never recover completely from the effects of them, and in a certain number of cases where the brain has been damaged other troubles may ensue later on, as in the case of the man we had in Abernethy Ward some two years ago and whom some of you may remember. He had a very serious injury to the brain, with fracture of the base of the skull, and lost an immense quantity of blood and was unconscious for three weeks. He ultimately made a satisfactory recovery, and I explained to him the necessity of being extremely careful about drink, and I think he was very careful and never disobeyed orders in this respect. A year after that, however, when apparently well, he began to have epileptic fits, and I have no doubt that they were a consequence of the injuries to his brain. So you must remember that for a long time after apparent recovery from serious injury to the brain, complications may ensue, and these may come on as much as a year after the original trouble. Then there is a woman who comes to see me sometimes now, who was in Stanley Ward rather more than a year ago with fracture of the base of her skull. She recovered. The fracture of the base of the skull was complicated by evidence of injury to the brain. She had severe concussion, and was semi-conscious for a long time. What does she complain of? She came three months after the accident saying that when in the street she was so disturbed by passing traffic that she became confused and would fall if she did not take hold of somebody. She said that any noise so upset her that she was liable to get giddy. At the end of rather more that a year these symptoms gradually passed away, but this will indicate to you that it may be a very long time—it may be a whole year—before the symptoms have entirely subsided.

I showed you a picture just now in which there was pressure on the brain from a clot between the dura mater and bone, and I explained to you that this accumulation of blood might cause the patient to suffer from "compression of the brain." This is a different thing from concussion. What are the symptoms of compression? The symptoms of compression of the brain are such as you might expect. When the brain is gradually compressed the patient gradually becomes paralysed. This is not merely insensibility. A person though insensible can move his limbs. The condition of "compression" results in a clinical state which we call "coma," and coma is more than insensibility. When coma is fully established the patient is completely paralysed in all his voluntary muscles: that is what characterises coma. Mere insensibility may last, as I have said, for a short time, or it may last even for week, but where there is compression of the whole brain mass the patient becomes "comatose." As he becomes comatose his voluntary muscles become gradually weaker and weaker, and ultimately they are paralysed. And this is not only true of his voluntary muscles but of his involuntary muscles as well. For as he becomes comatose one of the characteristic things noticed is that his breathing becomes "stertorous" or noisy. Why? What makes the noise? As he is breathing stertorously you see that his cheeks flap out, and if you could look at his soft palate you would see that it was pendulous, paralysed, and his cheeks and lips are paralysed. It is the vibration of these paralysed soft parts which makes the noise. If the compression persists and the brain becomes more and more compressed, ultimately the involuntary muscles will become paralysed also, and respiration, which is mainly an involuntary act, will slowly cease, and in that way the man will die. So you see compression of the brain is different in its symptoms from concussion. It is true that the one may merge into the other, that a person who has a torn brain may bleed to such an extent that as the bleeding continues blood may accumulate sufficiently to compress the brain, and so a person who starts with having concussion may end with the symptoms of compression. Then, the essential clinical condition of compression is that the patient is becoming paralysed. What evidence is there that he is becoming paralysed? His pulse is large and soft. Why? What does a large and soft pulse mean? It means a semi-paralysed artery. That patient's arterial wall is no longer in a state of tone. If

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you look at the patient's eyes you see that his pupils are dilated, and that they do not react to light. What does that tell you? That he has paralysis of his third nerve. Cast a ray of light on the cornea and there is no response or reflex. There is evidence therefore of paralysis. What you feel in his pulse and notice in his eye are evidences of the condition which is the essential condition of compression of the brain, namely paralysis. In itself it is nothing that the pupil does not contract. The importance is that it is an indication of the paralysis of the iris. In itself the fact that the pulse is full and slow does not mean much harm, but it is an indication that the arterial system is paralysed. The essential thing is the paralysis, and by the extension of that paralysis the patient ultimately dies.

Remember, therefore, that contusion of the brain or compression of the brain may either of them complicate fractures, and when a patient has a fracture of the skull and is suffering from symptoms of compression, you read that to mean that in some way or another this injury has not merely affected the bone, but that something else has happened, either the brain has been torn or there is a bleeding vessel between the dura mater and the bone, and yet this may not be the direct consequence of the fracture, for the injured brain may not necessarily have received its injury from the fractured bone itself, but from the same violence which broke the bone.

*July 27th*, 1908.

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**Artificial Feeding of Infants.**—Johnson considers that the increasing prevalence of artificial feeding is because every year fewer women either can or will nurse their infants. The principal factors in inability to do so are: (1) heredity—alcoholism in the grandparents or inability of the maternal grandmother to nurse the present mother in infancy; (2) the artificial life of the modern women. To these Johnson adds as at least a contributory, and one that physicians can help to control, the strenuous school life of girls to-day. He reminds us that in rearing a girl we are contending with a deeper and more mysterious problem than in raising a boy, and suggests the inadvisability of continuing a parallel curriculum for boys and girls.—*Journ. A.M.A.*, vol. li, No. 1.

# A LECTURE

ON

# SPRAINS.

Delivered at the Medical Graduates' College and Polyclinic.

By J. JACKSON CLARKE, M.B., F.R.C.S.,
Surgeon to the Amalgamated Hampstead and North-West London Hospitals, and to the Royal National Orthopædic Hospital.

GENTLEMEN,—I have chosen the somewhat homely subject of sprains for this afternoon because it is a very important one, and there is nothing that requires our attention more closely, in the first place for diagnosing whether there is but a slight injury in a case which the patient would call a sprain, or whether it is something more serious. And it is also important to know how to treat these simple injuries. Those who see most cases of sprain are not medical men, but such persons as are termed "football doctors" and the like, who attend football matches, and have a reputation for treating slight injuries; they may be blacksmiths or farriers, or almost anything. It is hardly right that such matters should be left to these amateurs. A considerable section of the population suffers nowadays from a want of fresh-air exercises, and as far as we medical men can conduce to it I think we should try to check these large groups of spectators and convert them into active agents, making people play outdoor games themselves rather than going as spectators to great tourneys. Sprain is, as I have said, a somewhat homely topic, but we may remember that one of the great founders of medicine, Galen, held the office of physician to the school of gladiators at Pergamon, and I have no doubt that an important section of his experience was gained in his attendance on athletes at that time.

What is a sprain? What shall we give as the definition of a sprain? It is rather difficult to define it, but we can give an example in the case of the ankle. It is an injury caused by the joint being bent in some direction in excess of what is usual to the particular individual; an excessive movement of the joint in any given direction. In the case of a sprain of the back, two or three or possibly more joints are involved. But as we see

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it commonly in the ankle, knee, wrist and shoulder, it is a single joint. And even in the back it may be only one joint which is involved. But having so defined it, it is not so well characterised a lesion as a fracture or dislocation. That excessive movement of the joint in any given direction may result in the rupture of a ligament of the joint, and that is a very common condition in severe sprain. But most sprains, the milder degrees of the injury, do not entail so much anatomical damage. Most sprains are, I think, a stretching of one of the annular ligaments, those thickenings of the deep fascia which restrain the tendons, or some injury to the synovial sheaths of the tendons themselves. Thus a sprain is an injury uncomplicated by any more serious lesion such as a fracture or a dislocation. The differential diagnosis of sprain from other injuries is the most anxious matter for a medical man. The "football doctor" does not trouble himself about this aspect of the matter because he does not know, for instance, that what may appear to be simply a sprain in the spine may have been a dislocation or fracture of the vertebræ, which has righted itself, and which at any moment may become a complete dislocation with pressure on the spinal cord; what appears to be a sprain of the ankle may be a fracture of the fibula, and if the patient is allowed to walk on it that may become at any moment a complete dislocation of the ankle outwards —Dupuytren's fracture as it is called, with a great amount of deformity. During the past week I have been seeing a case where the latter deformity had become persistent, although the patient was put under an anæsthetic and an attempt made to reduce it. The man thought he had a sprain and was walking home, and just before he arrived at his door the complete dislocation took place. So the differential diagnosis of sprain is important. Before I pass to the principles of treatment of sprains we will consider a little further the complete anatomical diagnosis of sprain. I mentioned just now ruptured ligament. How shall we know whether a ligament is ruptured, or whether it is merely a stretching of the anterior annular ligament of the ankle? One can generally tell something from the history. A short time ago a patient was sent to me for an injury to one knee. He could only walk with difficulty with the help of two sticks. The history was that at the bottom of a steep incline he had fallen from his bicycle, and when he tried to get up he found his leg, instead of being in the usual antero-posterior plane, was bent at a right angle outwards from the knee, and it was necessary to use some force in order to get it straight. He was a man in responsible employment, and had to ride his bicycle a good deal in the course of his work. He said that he could not lay up, and must get on his bicycle again in a few days. From the history I surmised that the internal ligament of the knee-joint had probably been torn, and on examination there was still some puffiness about the attachment of this ligament to the outer condyle, and any attempt to put the limb outwards again caused the patient to complain of pain. The injury was not followed by effusion into the knee-joint, or, if there had been any effusion into the joint, it had gone when I saw the patient. I instance this case, since it affords an example of the main principle of treatment of sprains nowadays: this principle is to let the patient use the joint after we have supported that joint in such a way that it will not be displaced again with ordinary care on the patient's part. In the case of an ankle-joint we do this by strapping; but in the case of a big joint like the knee I had to do something more than can be done by strapping. I oiled the limb and took a piece of household flannel, shaped it to the limb, and after soaking it in thin plaster-of-Paris, reapplied it, and in a few minutes I had a cast of the man's knee. The action of the knee-joint is mainly antero-posterior on an axis drawn through the back parts of each femoral condyle. So on the cast I marked with a bradawl these points, and drew on the cast a plan for the instrument-maker to make a steel support which would allow free action on that axis, and at the same time prevent any return of outward displacement by means of an internal knee-cap. The instrument-maker, having the cast as a guide, was able to send the instrument ready for use a few days afterwards, and the patient walked away with it on. He had come limping before, but when wearing the instrument he walked quite naturally, and began to ride his bicycle at once. Here, then, is one example of our modern views and methods of treating a sprain: Let the patient use the joint after we have supported it in such a manner that the displacement which caused

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the damage cannot recur. The ordinary sprain of the ankle, which is due to a sudden twist of the joint inwards or outwards, how shall we treat such a case when we see it, at once, before any swelling has set in? Most of you, no doubt, are in the habit of strapping the ankle for such a condition, and I have brought examples of the kinds of strapping which can be safely used. You know that the ordinary rubber plaster is very irritating to the skin, especially in hot weather, and after taking such strapping off you will probably find the patient has more or less dermatitis. And the stickiness of the plaster is so great that it may irritate the skin by dragging upon it as the limb is moved. Here is strapping made in sheets by Fitch & Ibbotson, chemists in Lower Seymour Street. It is not too adhesive, and it is not irritating. Seabury's zinc oxide plaster may be used, but it is more adhesive. If we are called to see a sprained ankle before swelling has come on, we should raise the limb so as to allow the venous blood to run out of it, and flex the ankle to a slightly acute angle. The limb is then held in the horizontal position, and four strips of two-inch strapping applied. The middle of the first strip is applied to the sole of the foot in front of the heel, the ends crossing over one another in front, just below the ankle out over the heel, to prevent the skin of the heel being rubbed there. Then a second piece is applied at the back of the joint, the ends crossing in front. Two or more other pieces are applied above and below the second piece, a notch being cut in the lowest to avoid chafing of the heel. A strip of more adhesive plaster may be put over all, but it is not to be allowed to touch the skin. Those pieces of two-inch strapping will be sufficient to support that ankle, and if the patient takes care to keep the foot raised when resting he can use it in walking. The strapping will require to be renewed in two or three days, and again four or five days after that, in all probability, and the ankle will prob ably be well in a fortnight. But if in such a case the patient is not seen until some hours after the accident, the ankle will be swollen, the patient will have allowed the foot to drop, and if treatment is deferred for, say, a fortnight, shrinking of the calf muscles will have occurred, and adhesions about the ankle-joint will be formed, and the patient, unless properly treated, may be lame for the rest of his life. In order to prevent shortening of the calf muscles we must, when strapping the ankle, take care that the foot is at a right angle with the leg. As soon as the ankle is strapped the patient must begin to walk, at first putting the foot down flat without bending the ankle, but soon he begins to move the ankle and to use the calf muscles. In strapping the ankle in the way I have described we make what is really an artificial accessory annular ligament, and I think that in the commoner cases of sprained ankle it is the annular ligament which is damaged by being over-stretched. In more severe cases I apply an additional piece of strapping. Suppose it is the outer lateral ligament that has been injured, I apply a two-inch strip of strapping under the foot and bring it up the outer side of the leg a certain distance, and then put on the four pieces already described. The foot is made a little more secure against the return of the displacement. In the case of the wrist it suffices to apply two pieces of strapping round the joint and encouraging the patient to use the fingers. As in other departments of medicine, so in every case of sprain we have to study each case on its own merits. I have already referred to an extreme case of complete rupture of one lateral ligament. In a case of less severe sprain of the knee we have first to exclude synovitis: when there is effusion into the synovial cavity, the joint requires a little more consideration. It is best to let the joint rest for a week, bandaging it comfortably over cotton-wool and resting the limb on a splint. In such cases I have the bandages taken off twice a day to bend the joint a little to prevent adhesions forming. Thus when synovitis occurs we must put the case into a different category from sprain of the part. Also, in the case of the knee, we must be careful not to overlook fractured patella—an error which is easily made, and also easily avoided. Sometimes after what has been thought to be a simple sprain of the knee or synovitis the patient finds he cannot walk, and one half of the patella may be five or six inches above the other. Another injury at the knee which requires to be carefully looked for before we decide that a case is merely one of sprain is separation of the quadriceps tendon above the upper edge of the patella. Another is separation of the *ligamentum patellæ* from the patella or from the tibia. All

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such more serious injuries have to be more carefully considered, and wherever there is any doubt, for instance in the hip-joint, and its neighbourhood, and in fact in any joint, we must have a skiagraph taken. We will suppose there is no doubt that the condition is merely a sprain, *i. e.* a temporary over-stretching of some of the tissues without any effusion into the synovial cavity; then we strap the knee-joint in much the same way that we strap the wrist or the ankle. But we must make a little extra provision for the sharp edges of the patella, and also for the delicate tissues in the hollow behind the knee. We put over the patella a pad of cotton-wool a little larger than the patella, and another piece into the hollow of the knee at the back, and begin to strap in much the same way as we have strapped the wrist, using circular pieces of strapping, one overlapping the other. Then the patient may be allowed as much movement of the knee as that dressing will allow.

Before leaving sprains I should like to mention one other point. I have only spoken of cases where we are called in to see the patient immediately after the accident. But suppose the doctor has not been sent for immediately and events have been allowed to run their own course, and considerable swelling has occurred in consequence. The patient has probably been sitting in a chair with the foot on the footstool, and swelling will be considerable at the end of twenty-four hours. In such a case we should still strap the limb and change the strapping at least once a day until all the swelling has subsided. In other respects the treatment will be the same.

Now to pass to other various injuries, such as laceration of muscles, one example of which, I think, is called "lawn-tennis leg." Of this injury I have had personal experience: it consists anatomically of a tearing of some fibres of the calf muscles. Another similar injury is called "rider's sprain," and is due to laceration of some fibres of the adductor longus, or some of the other adductors. There are also "cricket shoulder," and "cricket thigh." Cricket shoulder is an injury of the deltoid, or the long tendon of the biceps. Cricket thigh is a bowler's injury to some of the muscular tissue of the quadriceps extensor. These are very painful little injuries, and if they are treated in a wrong way it may be a very long time before they get well. Take the simple case of a superficial rupture of fibres of the gastrocnemei. The feeling is the same as if one had been struck across the calf with a cane or a piece of wire, and if the painful spot is palpated immediately after the pain is experienced a distinct depression from a quarter of an inch to half an inch in width is felt. Left to itself the spot may remain painful for a long time. It is a laceration of muscular tissue owing to sudden excessive action. It was, I believe, Mr. Wharton Hood who first showed that this injury is also best treated by strapping whilst the limb is held with the knee extended and with the ankle at a right angle with the leg. The strapping is applied round two thirds or three quarters of the circumference of the limb, beginning below and ending just above the seat of the injury, so that the damaged parts are supported as with a very light splint, and yet the muscle is allowed to act, and the formation of adhesions between the damaged and the sound portions of the muscle is prevented. Treated in this way the patient may resume his business the day after the injury, or even the same day without any serious effects, and a much quicker recovery follows than would have been the case had he been put to bed with a splint on. I have not said anything about massage in these cases, but all these cases will be assisted by a little common-sense rubbing in the shape of centripetal frictions beginning at the proximal side of the area of effusion. Wherever there is swelling begin at its upper edge, and if it is a liquid movable effusion it will rapidly disappear. Other ruptures of muscle include "rider's sprain," and to those who are accustomed to spend much time in the saddle this is apt to be a very serious thing. Men in country practice may know of cases of lives made miserable by the recurrent pain of this condition, which comes on when a man is attempting to jump, or anything which requires the strong action of the adductor muscles. In this rider's sprain sometimes the amount of laceration is considerable. There may be a hæmatoma as big as one's fist from a ruptured adductor. When the muscle s rent there is, of course, a good deal of effusion of blood. Those who have much experience of rider's sprain know that it should be treated on the same lines as sprains of other muscles and lacerations, that is to say, it should be supported by strapping beginning from well below

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the injury, and ending a little above the injury, and allowing the patient to move. As the strapping becomes loose replace it, and use gentle massage both through the strapping and when the strapping is being renewed. The patient is thus encouraged to use the muscles. It is scar tissue by binding the damaged to the healthier parts of the muscle which causes the pain on movement.

*Sprains of the elbow.*—"Tennis elbow," "golfer's elbow," etc., are often due to a slight separation of the muscular atrachments to the outer condyles caused by any rapid twisting of the wrist or sudden extension of the elbow. In these cases again support is required by means of strapping from the middle of the forearm up to the elbow-joint, the uppermost piece of the strapping being cut hollow in front so that it shall not impede flexion of the elbow. In this way we keep the patient free from any adhesions and free from any exceptional wasting of muscles. I need not go further into these types of injury. But just as the principle of treatment of simple sprain is to support the part and allow the patient to use it, so it is in these slight lacerations of muscles.

We have to be extremely careful in the matter of diagnosis. We carefully exclude severe injuries such as I have mentioned, injury in these tendons or rupture of the tendo Achillis, fracture of the patella, Pott's fracture, Colles's fracture and the like. But when we know that the patient suffers from none of these we encourage him in every possible way not to be afraid of his injury.

There is another class of case not quite comparable to a sprain, but which deserves mentioning in this category, and that is writer's cramp, or occupation spasm. I have seen a good many cases of it, and very troublesome ones they are. You see it in violin and violoncello players as well as in writers and draughtsmen, and I have no doubt it occurs in connection with other occupations too. I have had it to a slight extent myself when I have been making drawings for my books. It is not so much a fatigue of muscle which we have to do with, but it is essentially a fatigue of cerebral cells. I think, to begin with, it is the motor centres in the brain which are weary, and in such cases the great thing is to get the patient to rest as much as possible from the occupation which has caused the trouble, and then to use the same limb for bigger movements. When I have had it myself I have noticed the wasting of the muscles of the hand; but if I have taken a few days' holiday and gone on the river rowing; that has had a much better effect than resting the hand for a day or two in a sling. So I say you should substitute for the fine and extremely differentiated movements movements of larger amplitude, such as rowing. A less interesting form of exercise is by repeatedly pulling up a weight by means of pulleys in one's own room, but I prefer to take my exercise out of doors whenever possible. Unfortunately, many of the patients who suffer from writer's cramp are dependent for their living on their daily occupation, and such a condition is of the most serious import to them. But it is a cheap investment for such to spend what money they can on a holiday and use the limb for coarser movements and exercises.

There is another condition which I should like to mention here, and that is painful displacement of the coccyx. There is usually in such cases a history of a fall on the coccyx, which leaves that painful condition which we know as coccygodynia. Following such injury or sprain of the coccyx there may be a painful state lasting years, and at every action of the bowels there may be a considerable amount of suffering. Where, from such a slight injury, such a painful and miserable state ensues, I am very careful to look out for a constitutional cause for the persistence of the pain. Most of these patients I have seen have suffered from indigestion, or some of the precursors of gout or goutiness. So that the condition is not always a simple traumatic one in the case of coccygodynia. When we meet with that condition what ought we to do? First of all examine the coccyx. In a patient I saw recently with the condition the coccyx was absolutely ankylosed, and ankylosis had occurred in such a position that it made a definite prominence on the skin, as if the lady were trying to prove her descent from ancestors older than those who came over at the Norman Conquest. There was quite a prominence at the end of the sacrum, and it was so rigid that it seemed as if there had never been a joint there at all. In a case of that sort, where sprain is followed by complete ankylosis in a bad position, there is only one remedy. If it is painful the coccyx must be removed. But in the early days of the coccygodynia, while it can still be regarded as a sprain, one would naturally examine as to whether there is any displacement, and, if there is. to redress it. The coccyx may be displaced towards the lumen of the bowel, and then under an anæsthetic a little traction backwards will put it into its proper place, and the bend may subside. But wherever the pain makes the patient's life a burden it is not a difficult operation to remove the coccyx, but it has to be done with great care in one respect and that is to secure asepsis, both at the time of the operation and subsequently—a rather difficult task in this situation, but one which with care can be achieved. Just as in the cases of injury referred to above we have learnt to minimise the amount of fixation of the injured part and to encourage early movement and to use massage, so has it been with regard to certain fractures. The latter, however, I hope to deal with in a future lecture.

*July 27th*, 1908.

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# SOME OUT-PATIENT NOTES AND SKETCHES.

By THEODORE FISHER, M.D., F.R.C.P.,
Assistant Physician to the East London Hospital for Children, Shadwell.

Most of us, no doubt, possess brief notes of many cases. When we come to look at these notes some years after they were taken, all recollection of not a few of the cases to which they refer may prove to have passed away, and we probably wish that the records had been more full and definite.

Almost valueless though such incomplete records may be from the scientific standpoint they not uncommonly present features of interest, and it is in the belief that the following are not devoid of at least some interest that I bring them forward.

*A case of habit spasm.*—In 1904 a not unintelligent boy, æt. 11 years, said to be in the fourth standard at school, was taken to the Hospital for Sick Children at Bristol. He had his hands tucked into his clothes between his trousers and shirt, and at intervals of two or three minutes compressed the abdomen strongly, almost violently; at the same time he bowed the body forward over slightly flexed thighs, uttering as he did so a loud grunting noise. Possibly the movement might be called a salaam spasm, but it was suggestive of sudden attacks of pain within the abdomen, and this view of the case was heightened by the expression of his face during the spasm. Apparently, however, not only was he not suffering from intermitting abdominal pain, but the spasm did not appear to have originated in some passing ailment affecting the abdomen. The first abnormal movement had commenced about a month before, when he would suddenly seize the hair hanging over his forehead. He gradually abandoned this movement for that above described. The boy was sent by me to a neighbouring photographer to have a snap-shot photograph taken of him. Unfortunately this photographer appeared to think a good view of the boy's face essential so he gave him a chair to lean against or to sit upon, and thus failed to obtain a good representation of the curious doubling-up movement. Yet in the photograph (Fig. 1) it will be noticed that the boy is not only pressing on the abdomen but is also raising the left leg, and some idea may be gained of the wide range of movement which took place when he was standing up, and not in any way endeavouring to keep still to "have his likeness taken."

The boy was said by his mother to be naturally nervous, especially of being in the dark, but no history of fits or of nervous disease in the family could be obtained. I wished to admit him to the

Fig. 1.

hospital, but the mother desired that consent of the father should be obtained. Not seeing him again at the end of about a week I called at his home and found that he had obtained admission into a hospital nearer to where his parents lived. I spoke to the physician under whose care the boy had been admitted but he was ignorant of the fact that he had anything of the nature described in his wards. On inquiry at the hospital it appeared that the boy had been admitted by a resident for "chorea," but that the movements

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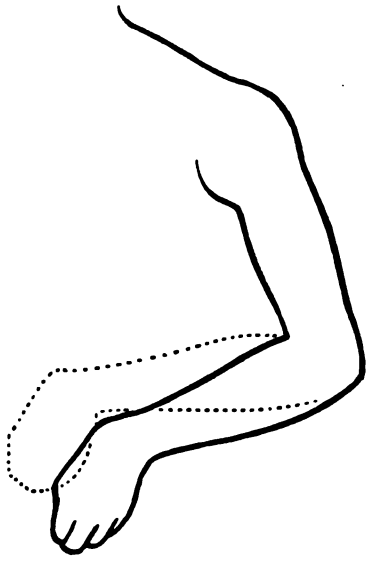
ceased soon after he was in bed, and did not return while he was in the hospital, although he was not kept in bed during a considerable part of the time.

*Localised spasm of doubtful origin.*—The next case is also one of spasm occurring in a boy, but one of lasting instead of temporary character. A boy, æt. 7 years, was seen five years ago, who was suffering from attacks of spasm of the left arm which were said to have started only six days before. They occurred at frequent intervals, there being twenty or thirty attacks or more a day. One attack occurred in the out-patient room. The elbow was semi-flexed, the fingers of the hand widely spread, and the whole arm raised until the fingers reached a point above the level of the top of the head. The fingers and wrist were then strongly flexed, the arm lowered to the side where short jerky movements of the forearm on the arm took place. Hurried rough sketches of the movements were made at the time, slightly elaborated copies of which have been reproduced here (see Figs. 2 and 3). The spasm occupied about half a minute. The boy appeared to attempt to restrain it and cried during and after its occurrence. Although rather pale and delicate-looking there appeared to be nothing definitely wrong with him. The possibility of the presence of a small tuberculous tumour was thought of, but nothing could be discovered to support this view. Spasms of the same character continued during the few weeks he was under observation. Recently his mother has been written to, who in reply gives the somewhat strange information that the left arm is no longer raised, but that the right arm and the right leg are raised at the time of the fits. The spasms also do not occur now as before, without loss of consciousness. The boy falls down, but whether before or after the arm and leg are raised the letter does not make quite clear. To sum up the leading points of this case there were at first spasms of the left arm and hand without loss of consciousness, and during the space of five years the spasms have spread to the right arm and leg while they no longer affect the left arm. There is also now loss of consciousness. In such a case one expects a local lesion to be present. But most local lesions would have ended fatally within five years. Even serious lesions like a tuberculous tumour, however, need not progress to a fatal ending. I have made a post-mortem examination on a case where a calcareous mass was present in the brain which appeared to be without doubt of tuberculous origin. Yet in the above case if focal tuberculosis were the cause of the spasms there would need to be two tumours. One tumour could scarcely affect the arm centre on one side and the arm and leg centre on the other. But whatever the cause may be one would think that localised spasms, occurring over a period of five years, must have an organic basis. Possibly it is of the nature of localised cerebral degeneration instead of the widespread

FIG. 2.

FIG. 3.



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degeneration of the brain sometimes seen in children.

*Paralysis of the sixth and seventh nerves due to a lesion on the pons Varolii.*—In the following case little need be said. Here a small tuberculous lesion was the cause of the first noticed signs of disease, and existed some months before the affection became more widespread and caused death. A girl, æt. 7 years, was seen by me on April 14th, 1902, in whom there was paralysis of the external rectus muscle of the left eye and paralysis of the left facial nerve (see Fig. 4). No anæsthesia

FIG. 4.

of the face could be detected. She presented no other symptoms until about the second week in July, when tuberculous meningitis developed and she died at home on July 23rd, more than three months after indications of paralysis of cranial nerves had made their appearance. The mother informed me of the child's death, and I took the opportunity of obtaining permission to perform a partial post-mortem examination. In the brain I found a small tuberculous mass not larger than a small pea in the pons Varolii situated very close to, if not in the actual site of, the nucleus of the sixth nerve. Destruction of the nucleus of the sixth nerve has several times been noticed to cause loss of power to move both eyes towards the side of the lesion. Unfortunately I did not examine for loss of power of movement of the internal rectus muscle of the right eye in this case. The photograph, however, shows the face turned very slightly to the right. Had there been weakness of the internal rectus muscle of the right eye one would have expected at least a slight involuntary turning of the head to the left. Possibly at the time the photograph was taken the small area of the pons Varolii affected by the small tubercular mass was just below the nucleus.

*Facial paralysis associated with paralysis of limbs on opposite side of the body.*—The following case presents some unusual features. A child, æt. 11 months, was seen three months after it had suffered from a convulsion which left behind paralysis of the right side of the face and the right arm and what at first sight appeared curious, not the right leg but the left leg. The paralysis of the face, it should be mentioned, was very definite and involved not only the lower half of the face, but the right eye and the right side of the forehead. When crying only the left side of the forehead was puckered and the left eye was partially closed.

Infantile hemiplegia is attributed by some pathologists to an encephalitis affecting the cortex, and it can be conceived to be possible that the leg centres might be affected on one side and the centres for the arm and face on the other, yet it is far more probable that the lesions of infantile paralysis were present in this case. Allowing, however, that the lesions in the spinal cord were the explanation of the paralysis of the limbs, the paralysis of the face remains to be accounted for. We may suppose that in addition to the foci of disease in the spinal cord there was a small area of inflammation in the pons Varolii affecting the nucleus of the facial nerve. Here again, however, we meet with difficulty. Sir William Gowers has recorded a case of facial paralysis, associated with infantile paralysis, attributed to disease of the nucleus, but the upper half of the face escaped. In this case, as has been mentioned above, the upper half of the face was markedly affected. The question arises, Does disease of the nucleus of the facial nerve invariably affect only the lower half of the face? It may, it is needless to remark, be argued that in this case the facial paralysis was due to disease of the middle ear. If that were so it would be a remarkable coincidence for a child to have a convulsion and after recovery from the convulsion be noticed to have paralysis of two

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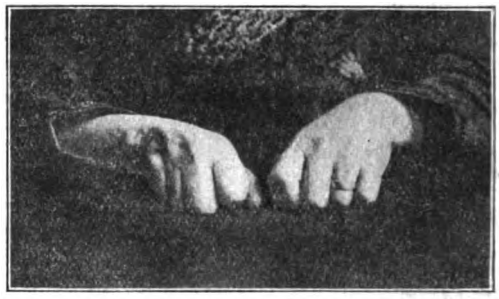
limbs and of the face, the paralysis of the limbs being due to disease of the spinal cord and of the face to disease of the middle ear. It may be interesting to add that the child is now twelve years of age. In answer to inquiry the mother writes to say that there is wasting of the whole of the right arm from the shoulder downwards. The weakness and wasting of the left leg are not so marked, and apparently affect mainly the thigh.

Of the face it is said it is "smaller on the right side." The boy is intelligent. Although only twelve years of age he is in the sixth standard at school.

The following somewhat similar case may be worthy of mention. A child, æt. 19 months, was seen, which nine months before had suffered from a convulsion. After the convulsion had passed off the child recognised its relatives, but the whole of the left side, including the face, was found to be paralysed. So far the case may appear to have been one of ordinary infantile hemiplegia, but there are features which suggest that it may have been of the nature of infantile paralysis. The left leg and thigh were colder than the opposite limb and the muscles flaccid. No knee-jerk could be obtained on the diseased side, although it was well-marked on the right side. The left arm appeared to have completely recovered, but a feature of considerable interest was the persistence of the facial paralysis. The notes say, "when the child yawned the mouth was drawn to the right and the left eye was only partially closed. The right side of the forehead also alone was puckered." To add to the complicated nature of this case the convulsions were said to have recently returned. According to the description given both sides of the body, including the weakened side of the face and the left leg, were affected by the fit. The occurrence of convulsions must probably be looked upon as largely a coincidence. The fact that the leg and face remained paralysed while the arm recovered is not very suggestive of a cerebral lesion. The flaccid condition of the left leg and the absence of knee-jerk also seems to point rather to disease of the spinal cord. It may be added that although facial paralysis is a rare association of anterior poliomyelitis, its persistence in cases of infantile hemiplegia is also uncommon, especially the persistence of weakness affecting the upper half of the face. It would have been interesting to have known the electrical reactions of the muscles in the case. These probably would have settled any doubt as to the situation of the cause of the paralysis. In the absence, however, of certainty upon such a point, the association of widespread paralysis of one side of the face with paralysis of the leg on the same side, while the arm did not present any sign of weakness, seems to be an occurrence sufficiently uncommon to merit brief notice.

*Wasting of one half of the hand.*—The illustration (Fig. 5) shows wasting of the ulnar half of the

FIG. 5.



right hand. Comparison of the fingers on the inner and outer halves of the hand gives some idea of the extent of the wasting. The muscles on the ulnar side of the forearm were also wasted. This somewhat curious condition, which was seen in a woman, æt. 60 years, possibly originated in an attack of infantile paralysis at the age of thirteen years. She stated that she had severe pain one night in the right forearm and in the morning the inner half of the hand was found to be paralysed. The two inner fingers never regained power of movements and the corresponding part of the hand wasted. At the time the patient was seen these fingers were not only extremely small compared with their fellows on the opposite hand but were rigidly flexed. The bones of the affected part of the hand, judging by their size, must have grown very little since the time of onset of the paralysis.

*July 27th*, 1908.

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# THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE, AND CLINICAL REVIEW.

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# THE TREATMENT OF TAPE-WORM.

By F. de HAVILLAND HALL, M.D., F.R.C.P.,
Physician to Westminster Hospital.

CASES of tape-worm occur so seldom in hospital practice that students have very little opportunity of watching the treatment. For instance, during the last ten years at the Westminster Hospital I can find reference to only two cases of tape-worm being admitted into the wards. I have, therefore, thought that a clinical note on a patient recently in the hospital under my care might be of use in directing attention to the method of treatment which should be employed with the object of getting rid of the unwelcome visitor.

A. N. W—, æt. 25 years, a clerk, was admitted into the Westminster Hospital a short time ago. About three or four years ago he noticed that his temper was becoming more irritable, and that though he was very hungry and took more food he did not increase in weight. About eight weeks later he discovered a few segments of tape-worm in the motions, so he consulted a medical man, who administered male fern. Many feet of the parasite were passed, but the head was not found. The symptoms continued, and ten to twelve weeks after the administration of the male fern segments of the worm were seen again. The drug has been given every three to four months with a similar result. One week the patient took the medicine twice. In all he states that he had been treated for tape-worm fifteen times.

On admission the patient was a well-nourished man and of good colour. His bowels were constipated, and his motions contained a few segments of tape-worm.

The patient was placed on liquid diet, milk and beef tea, one and a half pints of each, no solid food being allowed. Medicinally he was ordered:

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℞ Sodii bicarb. . . . gr. xx
Sodii sulphatis . . . ʒj
Spiritus chloroformi . . ♏xx
Aq. menthæ piperitæ ad . ʒj
*t.d.s.*

As a result of this treatment his bowels were freely moved. On the evening of the third day the patient had the following draught:

℞ Magnes. sulphatis . . ʒss
Tinct. jalapæ . . ʒj
Tinct. chloroformi co. . . ♏xx
Aq. ad . . . ʒiss

Early the following morning male fern was given in a mixture:

℞ Extracti filicis liquidi . . ʒj
Pulv. tragacanth. co. . . gr. xx
Spiritus chloroformi . . ♏x
Aq. menthæ piperitæ ad . ʒj

An hour later the dose was repeated, and an hour after the second dose the patient was given half an ounce of castor oil with a drachm of the tincture of jalap. As the bowels did not act very promptly an enema was administered, and this brought away many feet of tape-worm, and the head of a *Tænia mediocanellata* was found among the segments.

The brief notes of the case I have given will illustrate the points on which I wish to lay stress. In the first place, I would direct attention to the failure of the previous treatment of the patient. Fifteen attempts had been made to relieve the patient of his troublesome guest, but though many segments were expelled, the head remained attached to the bowel, and after the usual period of three months segments were again passed. In the second place, I would remark that the directions for treatment given in the text-books in common use are not sufficiently precise. The practitioner, who has probably not seen a case of tape-worm treated during his hospital career, will naturally be compelled to have recourse to his text-book to see what line of teatment should be employed when he is called upon to prescribe for a patient suffering from this complaint. If he refers to more than one text-book he will find great divergency both as regards the dose of male fern to be employed and the steps to prepare the patient before the administration. I have looked up the four text-books which I believe are mostly in vogue at the present time. The dose of the liquid extract of male fern in one book is given as 15 to 30 minims, in two as a drachm to a drachm and a half, and in the fourth the dose is stated to be two drachms. Then as to the method of preparing the patient one authority says that "in most cases it is sufficient for the patient to have no food after six or seven in the evening, and to take the anthelmintic before breakfast the next morning." Another only says that "it is advisable for the patient to fast several hours before taking it" (*i. e.* the anthelmintic). A third advises that the patient should "take only liquids, such as milk and beef tea for a day," and the fourth advises that for two days prior to the administration of the remedies "the patient should take a very light diet." Only one of the four writers mentions anything as to the necessity of medicinal treatment before the administration of the anthelmintic, and he says that the patient should "have the bowels moved occasionally by a saline cathartic."

Following these totally inadequate directions, it is no wonder that medical practitioners fail to treat cases of tape-worm successfully, and if at times the head is expelled it is more by luck than good management that it happens. In order to ensure success in the treatment of these cases several points must be borne in mind. In the first place, the intestine must be as empty as possible. In the second place the catarrhal condition of the intestines which so often exists in cases of tape-worm must be remedied, as if the worm be protected by the intestinal mucus the anthelmintic may not be successful. Thirdly, the advisability of giving the liquid extract of male fern in two doses: it has been suggested that the first dose causes the worm to relax the very firm hold it has upon the intestine, and the second dose kills it right out. And lastly, the administration of a brisk cathartic to bring away the worm and the male fern, so as to prevent any toxic effects on the patient from the latter.

All these indications are met by the following plan of treatment which I have found to answer in every case in which I have employed it. It may seem rather strict, but in the long run it will save much time and trouble.

For three days previous to the administration of the male fern the patient should be kept entirely on a liquid diet. I have found that a pint and a half of milk and a like quantity of beef tea answer very well. To promote a free action of the bowels and to favour the removal of mucus a

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mixture containing twenty grains of bicarbonate of sodium, a drachm of the sulphate of sodium, and twenty minims of spirit of chloroform in an ounce of peppermint water should be taken three times a day.

The night before the male fern is given the patient should have a draught containing half an ounce of sulphate of magnesium with a drachm of tincture of jalap, and twenty minims of the compound tincture of chloroform in an ounce of water. This should be repeated next morning at 7 o'clock if the previous dose has not operated. At 8 a.m. a drachm of the liquid extract of male fern made up in a mixture with a drachm of mucilage of tragacanth and a drachm of the syrup of ginger in an ounce of chloroform water. At 9 a.m. this dose should be repeated. At 11 a.m. half an ounce of castor oil with a drachm of tincture of jalap should be given, and if the bowels do not act within an hour an enema of a pint and a half or two pints of soapy water should be administered. The motions should be carefully examined to find the head, and if the above treatment has been faithfully carried out it may be sought for with confidence. It is desirable to keep the patient in bed for two or three hours after the bowels have acted, as the male fern may cause faintness. The line of treatment I have depicted above was originally described by the late Dr. Leslie Ogilvie, and it is to prevent an excellent method from falling into oblivion that I have been induced to pen this communication.

*August 3rd*, 1908.

**Local Treatment of Acute Inflammations of the Throat from the Standpoint of Pathology.**—J. L. Goodale says that in a commencing tonsillitis antiseptic applications may perhaps be used with benefit with a view of aborting the attack, but if this initial procedure does not sterilise the parts, and the usual cryptic deposits appear with systemic involvement, further applications of antiseptics may be not only useless but even harmful. They may diminish the number of bacteria in the crypts which are generating toxin, and thus prolong the period required for the formation of the requisite amount of anti-bodies, and they may exercise destructive action on the tissue cells and phagocytic leucocytes of the host. —*Med. Record*, vol. lxxiv, No. 2.

# TWO CLINICAL LECTURES

ON

# INJURIES TO THE HEAD.

Delivered at St. Bartholomew's Hospital.

By ANTHONY A. BOWLBY, C.M.G., F.R.C.S.,
Surgeon to the Hospital.

## Lecture II.

Gentlemen,—I was speaking last week about the symptoms of concussion of the brain and compression of the brain. I want to-day to direct your attention more particularly to the compression of brain which is due to hæmorrhage between the dura mater and the bone. And you will see at once that by observing the symptoms of such a case before the whole of the brain mass becomes compressed, you are able to make a diagnosis when the pressure, which is becoming dangerous, and which will become more dangerous, is yet in progress. The fact that these specimens here are in the museum will indicate to you that this may be a fatal lesion. And that specimen shows particularly well the crack of the parietal bone and the tear in the middle meningeal artery, and here is another specimen which shows the blood-clot which is formed between the dura mater and the bone in such a case. In these cases there is often a gradual onset of symptoms, and as a portion of the brain becomes compressed, there is partial paralysis before the onset of symptoms of compression of the whole brain mass, to which we were alluding last lecture when I was speaking to you of the symptoms of compression of the brain as a whole. I will read you two typical cases which were described by Percival Pott here a great many years ago, but those descriptions are just as typical of this condition to-day as they were at the moment this was written. "A porter of the water-side was knocked down by a blow from a hook at the end of the tackle belonging to a crane. He was senseless for nearly half an hour, but after that was so well as to be able to walk home. Next morning he lost his sight and by the evening his speech and faculty of walking were lost, and in this state he was brought to the hospital. He was let blood and his bowels thoroughly emptied, and

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I intended if this evacuation did not materially serve him, to have examined the state of that part of the bone whereon the blow was received. But that night he died. Upon examining the head, a piece of the inner table of the parietal bone of about one and a half inches in length and not quite so broad was found having a quantity of blood between it and the surface of the dura mater." That man, you see, had symptoms which came on very slowly, and it was twenty-four hours after the injury that he became unable to walk. You will remember that he walked home immediately after the accident, and this indicated that he had no very severe injury to the brain itself. Ultimately he died of compression of the brain. Here is another case, and in this the patient's life was saved by timely interference: "A whitelayer's labourer fell from a scaffold and broke one arm and one thigh, and was brought to the hospital about two hours afterwards in a state of stupidity. When his arm and thigh were put to rights his head was examined, but no mark of mischief was discovered. He was bled freely . . . on each day for four days, but he continued in the same state. On the fifth day a small tumour of the bone on the right side of his head was found. The scalp was removed and the bone being found bare it was immediately perforated. The perforation made way for a large discharge of blood which had been contained between the dura mater and the skull. On the second day from this operation he remained the same; blood was drawn from some part of him and the discharge continued large and free through the opening which was made in the bone. On the fourth day, having taken laxative medicine, he was better, on the fifth he became sensible, and from that time he had no difficulty or trouble and recovered." Notice that in this case the bleeding was still more slow. This man was alive at the end of four or five days. And notice another thing which is mentioned in this case, which was not mentioned in the first one. There was noticed a swelling of the side of the head which gradually came there. What was that swelling? It was due to the escape of blood through the crack or fissure in the bone, so that the blood accumulated not only between the dura mater and bone, but also underneath the pericranium. It stripped that up and filled up more or less the temporal fossa. And that is one of the symptoms you must look for in a case of hæmorrhage from the meningeal artery with a crack in the parietal bone. But I want you to observe that in the first case the man was able to walk home, and that in the second case the man was stupid. He had concussion of the brain as well. Unfortunately there are many of these cases in which the patient has two injuries, that is, a fracture of the skull with laceration of one of the arteries, and bleeding between the dura mater and bone, and also in addition to that, he has a contusion of the brain and suffers from the symptoms of concussion. A few months ago a man was admitted here, having fallen from a scaffold. He was suffering from symptoms of concussion and was insensible. But after he had been in the hospital a few hours it was noticed that whilst he could move one arm and leg, the other arm and leg were becoming paralysed, and soon became completely paralysed. That indicated that one part of his brain was suffering more than the rest. Compression of the brain as a whole, as I told you, gives rise to paralysis and a condition of coma, but compression of a part of the brain will give rise to corresponding paralysis of a part of the body. The usual site of hæmorrhage between dura mater and bone is on one side of the skull, in the temporal region, and it follows that the pressure of this clot comes to bear most upon the Rolandic area, and consequently, as in Pott's case, the man may lose power of speech, and may gradually lose power in the arm and leg on the opposite side of his body from where the injury to the head has been. So what you look out for are the symptoms of paralysis due to local compression of a particular area of brain. In this case we operated and found a fissured fracture of his skull. We took out a portion of bone and found a large extravasation of blood between the dura mater and the bone. We cleared that out, but in spite of it the man did not improve. Unlike the case of Pott's which I described to you, after a day or two he was no better, then gradually he became worse and died. And then at the autopsy we found that this, as is so often the case, was an instance in which the patient had had two injuries, a fractured skull with a hæmorrhage between the dura mater and bone, and also an extensive laceration of the brain, and it was this laceration of the brain which gave rise to hæmorrhage at the base of his brain and ultimately brought about his death. It was

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the brain injury which killed him; and unfortunately that is often the case.

So much for local compression and the gradual onset of symptoms. But there is another class of case in which the injury is an injury to the brain alone, and the brain bleeds. I was speaking to you last week about the symptoms of contusion of the brain, and I was alluding to the condition of the patient which indicates that he has this injury. Let us go a step further. In a certain number of cases there is evidence that a particular part of his brain is torn. I came to the hospital to see a man who had been admitted with concussion of his brain, and soon after admission he began to have convulsive movements of one arm and leg, terminating in epileptiform fits. You notice that this was just the reverse of the condition of simple compression of the uninjured Rolandic area by a blood-clot, resulting in paralysis of an arm and leg. Here was a condition in which instead of there being paralysis there were violent convulsive movements of an arm and leg. And one had to ask oneself what was the explanation of that? What had happened in this man's head? It was almost certain that in this case there had been an injury to the corresponding part of the brain, and in this case it was not thought there was bleeding between the dura mater and bone, but that the injured part of the brain was the part which was bleeding. So I operated upon this patient, and we found nothing between dura mater and bone, but on opening the dura mater there was found a large quantity of fluid blood and clot, and for the time being the patient was relieved and the spasm ceased, and the insensibility became less marked. But that patient also ultimately died. Both these patients died because the injury to the brain was so extensive that the bleeding continued, and the powers of the brain remained in abeyance, and ultimately, after being in a state of coma, both these patients died.

Besides blood compressing the brain there may be another fluid causing compression, namely, pus. In every book on surgery which you read you will find allusions to what has been described as Pott's "puffy swelling." Pott described how, in a certain number of cases of injury to the skull, there occurred suppuration, and he pointed out how in certain of these cases there was an alteration in the bone, and how a swelling occurred in the head. And his descriptions have been copied and alluded to from time to time up to the present day, and you will still find that the descriptions in text-books refer to what Pott originally wrote. Let me say to you that what we are now going to speak of is injury to a bone causing acute septic osteitis. We are all rather accustomed to associate acute osteitis and periostitis with long bones and with children. If you look in our Museum you will see many specimens to illustrate acute periostitis of the tibia or of the femur, but we do not so often talk of acute periostitis or osteitis of the skull. And yet this is a very real thing, and, unlike the acute periostitis with which we are so familiar in the long bones in children, this lesion is just as likely to occur in the adult. It follows either a mere blow, without any external wound, or more often a blow causing a wound which suppurates and results in an extension of sepsis to the bone itself. Now, acute osteitis of any part of the body is very liable to terminate in death of the bone or necrosis. But in this case of course you must remember that on the other side of the necrosed bone there is dura mater, and, whereas in the leg or thigh the pus may accumulate on either side of the periosteum, or may only strip up the periosteum or a muscle from around the inflamed bone, here you have a space where if the pus collects it will cause pressure on the brain underneath, as surely as blood-clot will.

With that preliminary let me now read you this description: "If there be neither fissure nor fracture of the skull, and if the scalp be neither considerably bruised nor wounded, the mischief is seldom discovered or attended to for some few days." Notice that, because unlike a case of hæmorrhage or injury to the brain, there are no symptoms until the osteitis has had time to be established. In a case of concussion or hæmorrhage symptoms come on practically immediately. "The first symptom is generally pain in the part hit by the blow; and this pain soon extends all over the head, and is attended with a languor or dejection of strength, an inclination to vomit, . . . quick and hard pulse, and an incapacity for sleeping quietly. A day or two after this attack begins, the stricken part swells and becomes puffy and tender, neither does the tumour rise to any considerable height or spread to any extent. If this tumid part of the skull be divided, the peri-

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cranium will be found of a darkish hue, and either detached or easily separated from the skull, between each of which will be found a quantity of dark coloured ichor. If the disorder has made such progress that the pericranium is separated from the skull the latter will be found somewhat altered in colour from healthy bone. From this time the symptoms generally will advance more hastily, the fever increases, the skin becomes hotter, the pulse quicker, and harder. The patient will be more disturbed and the restlessness will be more fatiguing, and to these are generally added irregular rigors."

That is a fairly accurate description of acute periostitis and osteitis of the bones of the skull terminating in pyæmia, as far as the general condition of the patient is concerned and terminating in meningitis or in abscess of the brain as far as the local conditions are concerned. You can easily understand, then, how when this pus accumulates the patient gradually suffers from the symptoms of compression of the brain. But you see in this case the symptoms of compression of the brain are preceded by the symptoms of sepsis.

Pott describes a case, that of a fireman, in whom there was a contused wound of the head, and on the third day this man began to be ill, on the fifth day worse, and continuing to get worse he died in a state of coma, and it was found that he had suppuration over the surface of his dura mater.

These are cases, then, in which the symptoms come on after a very considerable interval of time. In these patients the symptoms of local compression are preceded by the general symptoms of sepsis, and consequently there ought not to be very much difficulty in diagnosing the condition of affairs before the matter has had time to extend through the dura mater and set up diffuse meningitis, or possibly abscess of the brain.

Now let us come to the next point, suppuration of the brain itself. This may result either from the direct injury of the brain or from extension of inflammation to it. In the case of that child whose case I related to you last week there was a suppurating brain though there was not an abscess of the brain. She had had a compound comminuted fracture of her skull, which had terminated in suppuration of the brain on its surface and ultimately in meningitis at the base of the brain, and consequently in the death of the patient. But an abscess of the brain is a condition which may ensue a considerable length of time after an injury. And here I show you specimens which illustrate how late some of these abscesses may appear. Here is one in which the trouble affected the frontal lobe. That was from a woman, æt. 26 years. The abscess appeared in consequence of a fracture produced by a blow on the head. Four days after admission there was pyrexia. The temperature remained high. A month after the injury the woman was drowsy, and paralysis of the right arm appeared, also ataxia, and paresis of the right side of the face. She was trephined, and pus found in the diploë of the bone. That was a case, therefore, like one we have been describing. She had an injury to her head, acute septic osteitis, and inflammation had spread from the bone to the membrane and thence to the brain. Later, convulsions occurred and unconsciousness, and drowsiness, followed by rigidity of the right arm. There was coma and stertorous breathing, and on the seventh day after the trephining she died. That is a typical case which illustrates how late after an injury of the skull an abscess may arise. This woman was in the hospital, and it was a month after the injury that the symptoms of abscess began to develop. Therefore you see this is a still later lesion than the accumulation of pus between the dura mater and bone. Here is another specimen, a portion of the cerebrum of a child. And this is a very similar history in many ways. In this case the patient was a child, æt. 4 years, who had an extensive scalp wound on the right side, followed by suppuration and exposure of the cranium. A month after the injury, whilst all seemed to be proceeding favourably, the child was seized with convulsions, which were followed by partial paralysis and insensibility. By means of the trephine a small quantity of matter was let out between the skull and the dura mater, but without relief, and the child died three days after the convulsions began. Here, again, there was injury to the bone, sepsis, extension of inflammation to the dura mater, the spreading of it through the dura mater rather than accumulation of pus between the dura mater and bone, and the development of abscess in the brain. And I want you to remember this next specimen as well,

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because it is one of an abscess of the cerebellum. In that case the patient had a fracture of the base of the skull. One is a little liable to forget that fractures of the middle fossa of the base of the skull, and generally fractures of the anterior fossa also, are "compound" fractures. Fracture of the middle fossa of the base of the skull, in a large number of cases, passes through the temporal bone, across the tympanum, tearing the lining membrane of the tympanum and the membrana tympani. Under those circumstances, of course, with the membrana tympani torn, you have a compound fracture. You are all familiar with the fact that suppuration in the middle ear due to old septic disease of the middle ear is very liable to be associated with extension of inflammation to the brain or its meninges. We are liable to forget that the same thing may happen with fracture in this region, and that fractures of the skull and associated sepsis are not always fractures of the vertex, but may be fractures of the base also.

Here is a specimen from a man, æt. 43 years, who fell from a cart on to his head, sustaining a fracture of the base of the skull. On admission he could answer questions. There was hæmorrhage from the left ear and left nostril. Three days after the accident there was a discharge from his ear, which became purulent, and he had suppuration of his fracture. One might have written exactly the same of a case of fracture of the tibia, you might say: "Three days after being put up in splints suppuration began, and the tibia became inflamed and necrosed." That is a common enough story. But this is the same thing in the head, and you must always try to keep before your minds that the same things which may happen in the bones in the extremities may happen in bones of the head. In this case, three days after the accident, the discharge from the ear became purulent and the patient died a fortnight later. That man died of abscess in his brain nearly three weeks from the accident. You will see, then, that abscess of the brain following fracture of the skull or contusion of the brain with sepsis is a late lesion.

Now let us put the brain complications in something like their order of time. Concussion of course produces immediate results. A person who has concussion of the brain is immediately affected according to the extent of his injury. A hæmorrhage between the dura mater and bone will come on gradually in course of a few hours, or at any rate within, ordinarily, twelve hours. A patient who has an injury to his skull followed by septic osteitis will have symptoms of sepsis within about a week. He will have symptoms indicating the presence of pus inside his skull in about a week to twelve days. A person who has abscess in the brain resulting from a fracture will very likely have symptoms pointing to sepsis for a fortnight or a month or more before the symptoms of the abscess appear. I do not propose to go at the present time into all the symptoms of abscess of the brain—it would take a lecture by itself. But I want you to remember that the symptoms due to abscess in a part of the brain which is not associated with some particular movement whereby the situation of the abscess can be localised, may not always lead you to suspect the true nature of the trouble, though there may be a considerable quantity of pus. The frontal lobes seem to be capable of accommodating more pus than any other part of the brain, and abscesses in the frontal lobes are consequently much more difficult of diagnosis, and the symptoms much more latent, than those of abscess in any other part of the brain.

I should be giving an incomplete lecture on this subject if I were to omit to mention that in consequence of injuries to the brain associated with suppurating wounds there may be ultimately a protrusion of the brain which is talked of as "hernia cerebri," that is a hernia or protrusion of the brain at the seat of a suppurating lesion. The hernia consists of inflamed and softened brain. It is pushed out partly by reason of the swelling of the inflamed cerebral tissue, but it is also pushed out by the constant pressure of the pulsating brain, and you must clearly understand this in order to know what treatment to apply. You know that any swelling in connection with the brain pulsates, that is to say it receives transmitted pulsation from the very vascular brain below. This pulsation of the brain will, in a case of hernia cerebri, tend to cause extrusion of a considerable quantity of brain. It is this more than the mere swelling of the inflamed brain which actually causes the hernia. The brain comes out partly because it swells, but mainly because of the pulsation of the brain beneath it, and, consequently, while you treat such a condition by any sort of antiseptic

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dressing which you like, you must remember that a certain amount of pressure support by means of a suitable bandage or dressing will tend to prevent an undue extrusion of brain. So that in many cases of hernia cerebri the extent to which the protrusion is permitted depends to a great extent upon the application of dressings. You must not suppose that when a person has a hernia cerebri he has a condition which is irremediable. If there is at the base of it some condition like necrosis of a portion of bone then of course it becomes more serious. But in fracture of the skull with tear of membranes and a lacerated brain, the fact that there is protrusion of brain at the end of a week or two does not mean that the patient has a lesion from which he cannot recover. In my experience a considerable number of patients do get well.

*August 3rd*, 1908.

**Arterio-sclerosis.**—Lancereaux protests against the assumption that arterio-sclerosis is an affection of old age. He has observed it frequently between thirty and forty, and has noticed that its course is more rapid in the comparatively young. The two main factors are gout and lead poisoning. Tobacco, liquor, and alimentary toxæmia have long been over-estimated, he thinks, as factors in arterio-sclerosis. They act only in case of a tendency to gout or lead poisoning. The practical result of his views is that members of families which display a tendency to gout should be treated early, seeking to modify that condition of the nervous system which predisposes to arterio-sclerosis. He thinks that he has done good work in this line, although its very nature prevents conclusive testimony. He prescribes potassium iodide, from 1 to 3 grm. (15 to 45 grains) in twenty-four hours, continuing this for months and years, with intervals of a week each month. He orders at the same time a drastic purgative to promote the elimination of the iodide. Hypertrophy of the heart with high arterial tension and nocturnal polyuria with lesser density of the urine are signs of diffuse arterio-sclerosis. Brine baths, alcohol rubs and tepid douches, by soothing the nervous system, favour the elimination of waste. The practitioner should seek to ward off the impending uræmia, asystole or cerebral insufficiency, and treat them with the greatest energy if already developed. — *Journ. A. M. A.*, vol. li, No. 2.

# A CLINICAL LECTURE
ON
# CONSTIPATION.

Delivered at Guy's Hospital.

By ARTHUR F. HERTZ, M.A., M.D.Oxon., M.R.C.P.,
Assistant Physician, Guy's Hospital.

GENTLEMEN,—In discussing constipation the first thing to consider is exactly what is to be understood by the term. A person is generally considered to be constipated when his bowels are not opened regularly every day. But there are many perfectly healthy people whose bowels only act on alternate days, and there are some whose bowels are opened even much less frequently than that. Dr. Morton tells me that, when he was working in an Edinburgh dispensary, a large number of old women who came to him had their bowels opened only once every four or five weeks, and when he gave them a purgative and it resulted in their bowels acting before the expected time, they came back on the next occasion furious at the result. But for practical purposes I think we may take it that anybody whose bowels are not opened at any rate once in forty-eight hours should be considered to be constipated.

It is, however, important to remember that it is possible to be constipated in spite of having the bowels opened daily. Thus it is necessary to inquire whether the amount excreted every day corresponds in amount with the residue which the food taken during the previous day should leave. Moreover, the passage of very hard and dry fæces, causing painful defæcation, even if this occurs daily, shows that there is undue delay in the alimentary canal resulting in an abnormal amount of fluid being absorbed. It is even possible for a person whose bowels are opened every day to be so extremely constipated, if an insufficient amount of fæces is excreted, that serious results may follow. In a well-known case of Nothnagel's, the patient did not recognise that he was constipated, as his bowels had always been opened every day, but he died from peritonitis following the perforation of a stercoral ulcer, because a small proportion of what should have been excreted was retained each day, so that an enormous quantity accumulated in the

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colon. It is possible to bring together all classes of cases by regarding constipation as a condition in which none of the residue of a given meal is excreted during the ensuing three days. Most people excrete a little of it already in the next twenty-four hours, and one can fairly say that an individual who does not excrete any of it in the following three days is constipated. In doubtful cases in which it is thought that a patient may be constipated, although his bowels are opened every day, two or three charcoal lozenges should be given one morning with breakfast. If none of the fæces passed during the next three days are coloured black, the patient may be considered constipated.

In order that the bowels may act regularly, that is to say in order to avoid constipation, the passage of the food through the alimentary canal should in the first place be at the normal rate, and secondly when the fæces have reached the rectum they should be excreted normally. There are two great classes of constipation : those cases which are due to the slow passage through the alimentary canal, and those which are due to inability to defæcate completely.* I will briefly go over with you the physiology of these two processes, which must be understood before the causes and the appropriate treatment of constipation can be profitably discussed.

It is possible by means of the X rays, after having given an individual two ounces of bismuth oxychloride in bread and milk, to trace the food through the alimentary canal. This diagram which I show you indicates the average normal passage of food through the intestines (Fig. 1). Four hours after a meal is taken there is a small shadow seen in the cæcum. Two hours later the shadow reaches the top of the ascending colon. In another hour it will have got half-way across the transverse colon. Ten hours after the meal is taken it will have reached the splenic flexure, and in another couple of hours it will be some distance down the descending colon. In twenty-four hours, if the bowels have not been opened, the whole colon will be visible. One can observe the arrival of the contents of the small intestine at the cæcum by simpler means than the X rays. For example, if a person takes an ordinary breakfast and one listens with a stethoscope over the cæcum, nothing is heard until four hours have elapsed, when one hears bubbling sounds which indicate that the fluid contents of the ileum are entering the previously empty cæcum. One can trace the same thing by

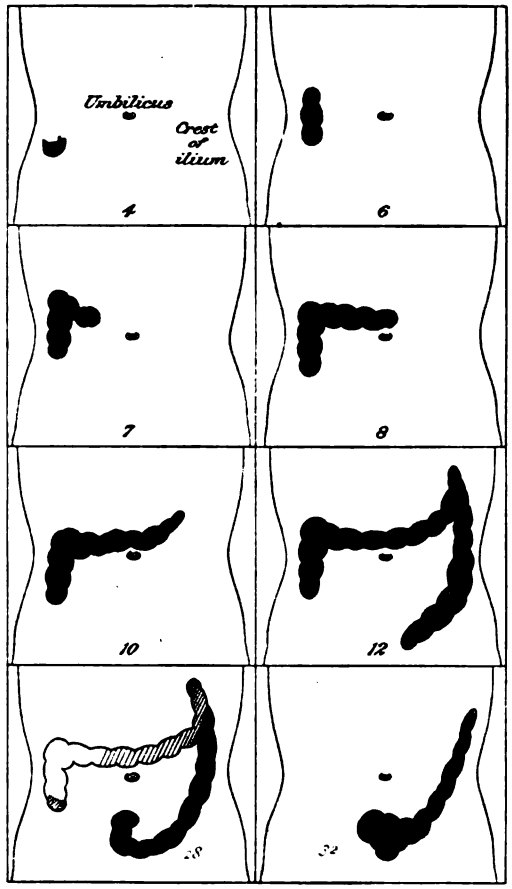


FIG. 1.—Series of colon skiagrams in a normal individual. The numbers represent the hours after a bismuth breakfast was taken.

means of percussion. Until four hours after breakfast the cæcum is always resonant to percussion; it gradually becomes dull later in the day as the contents of the small intestine reaches it, especially if the individual is examined in the vertical position. By palpation the changes in amount and consistency of the cæcal contents can also be recognised.

We now come to the question of defæcation. The desire to evacuate occurs as soon as a certain amount of fæces has collected in the rectum,

* Constipation is generally defined as a condition in which the passage through the alimentary canal is abnormally slow. The term would, therefore, hardly include the second class of case, which might be called "*dyschesia*" (δύς, difficult; χέζω, I go to stool), a word first introduced by Robert Barnes to denote painful defæcation.

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which is normally almost empty. The act of defæcation begins with the voluntary contraction of the diaphragm and the abdominal muscles. By these means the intra-abdominal pressure is greatly increased, and the fæces in the descending colon move towards the point of least resistance, that is to say, the anus. The rectum thus becomes distended and the normally empty anal canal now contains some fæces. These two factors give rise to the reflex act of defæcation, which consists in the peristaltic contraction of the whole of the colon and the cæcum, and not only of the descending colon, as formerly taught. The internal and external sphincters of the anus are simultaneously relaxed. There is thus a movement of the entire contents of the large intestine towards the anus, which is most marked in the distal part, so that normally all the colon below the splenic flexure empties itself completely. The process is seen in Fig. 2, which also shows the effect of the descent of the diaphragm on the shape and position of the colon. The voluntary contractions of the diaphragm and abdominal muscles are generally maintained throughout the act of defæcation, so as to increase the effect of the involuntary peristalsis of the colon.

FIG. 2.—Normal defæcation. A. 10.30 a.m., immediately before defæcation. B. 10.35 a.m., immediately after defæcation. C. 12.20 p.m.

We now come to the classification of cases of constipation. The list which I have written up (see the end of this paper) looks a long one, but it is not generally difficult to decide to which class a given case belongs, because one can recognise the cause in the majority of instances simply from the history and by a thorough general examination of the patient. The first of the two groups (I), constipation proper, contains those cases which are due to the slow passage through the intestines, and the second, "dyschezia" (II), those in which defæcation is abnormal. One can separate these two classes from each other by rectal examination, which should always be made in a severe case of constipation that is not cured by ordinary simple means. The descending colon, sigmoid flexure and rectum should be emptied by defæcation. So if defæcation is normal and there is merely a slow passage through the intestines, the rectum will be found to be almost empty. If the passage through the alimentary canal is normal, but the act of defæcation is inefficient, examination will show that the rectum is distended with fæces, and perhaps even that the anal canal contains fæces.

The first class can be further subdivided into (A) cases due to deficient motor activity, and (B) those due to obstruction. Here again there is a rough general way of distinguishing between these two groups. Where the constipation is due to deficient motor activity there will be no colic, peristalsis being less powerful than it should be. But if there is something obstructing the passage of the material down the intestine, the part of the intestine above the obstruction will be abnormally active, as can be seen by means of the X rays, the shadow of a bismuth meal passing down abnormally rapidly as far as the obstruction. There is thus over-activity of the intestinal muscles, which are trying to overcome the obstruction, and this may cause colic. There is also likely to be a certain amount of flatulence in these cases, because of the retention of gas behind the obstruction.

Constipation due to deficient motor activity may in the first place (1) be due to abnormal intestinal musculature. This is probably the cause of the constitutional constipation, occurring in people whose bowels from birth have acted sluggishly. One finds whole families suffering from the same condition. Deficient activity of the intestinal muscle may be due to anæmia, particularly chlorosis. Almost every chlorotic girl is constipated, and the constipation is due to the same sort of change which gives rise to her cardiac troubles. The heart is often dilated owing to the poor blood which feeds

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its muscle. Similarly there is an insufficient supply of blood to the intestinal musculature, so that it becomes inefficient. In the cachexia of tuberculosis and of cancer one meets with the same thing. A large majority of old people are constipated, owing probably to the gradually increasing weakness of the intestinal muscle, which undergoes the same sort of degeneration as other organs. As an example of this class of constipation I show you these tracings from a case of chlorosis. A bismuth breakfast was given at 9.15 a.m. on August 8th. The cæcum was reached in four and a half hours (Fig. 3, *a*), and the middle of the transverse colon two hours later (Fig. 3, *b*), so that up to this point the rate of progress was normal, but in the

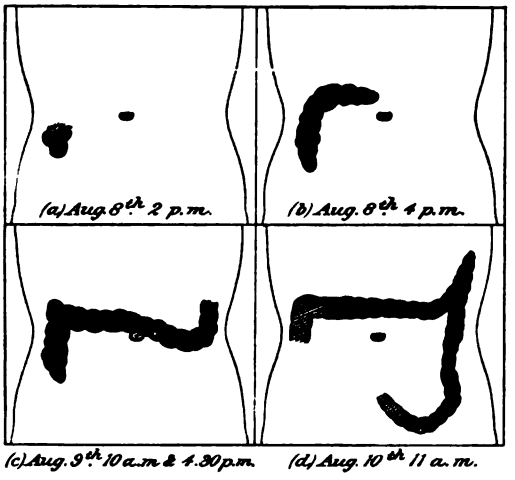


FIG. 3.—Constipation due to chlorosis. Bismuth meal at 9.15 a.m. on August 8th.

next twenty-four hours an advance of only three inches was made (Fig. 3, *c*). Thus the delay was only in the distal part of the colon. This case can be taken as a type of the majority of cases of constipation due to slow passage through the intestines, as there is hardly ever constipation of the small intestine, and also rarely of the ascending colon and first part of the transverse colon. The reason is that the contents in these parts of the intestine are fluid, so that even if the intestinal muscle is abnormally weak, the work to be done is so light that it has no difficulty in doing it. It is only in the distal half of the transverse colon and in the parts nearer the rectum, where the contents are normally solid, so that the work to be done is harder, that the delay is noticed.

The next group of cases (2) is that in which the deficient motor activity is due to defects in the nervous system. These we can divide into organic diseases, such as tabes, and functional diseases, such as neurasthenia, hypochondriasis and melancholia; probably the constipation of old age is partly due to this cause also. One of the most frequent causes of constipation is neurasthenia, the deficient intestinal activity being due to depression of the nervous system. The normal stimuli to intestinal peristalsis are, first, the mechanical irritation produced by the undigested residue of the food, and secondly, the chemical stimulus produced by such substances as organic acids, sugar, and oil. When the nervous system is depressed, the residue of the food does not produce sufficient stimulation of peristalsis. Here

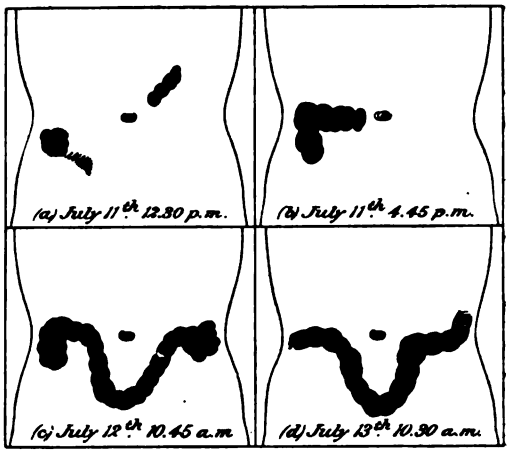


FIG. 4.—Constipation due to neurasthenia. Bismuth breakfast at 8 a.m. on July 11th.

is an example of constipation due to neurasthenia (Fig. 4). The delay is of the same type as in chlorosis, as the bismuth reached the cæcum and the transverse colon in the normal time, the passage through the transverse and descending colon being abnormally slow. When by suitable treatment the neurasthenia is cured, the constipation also disappears. Here are tracings (Fig. 5) from an interesting case, that of a medical man with hypochondriasis. He was 40 years old, and had spent the last twenty years of his life thinking about his inside, examining his excreta, and palpating his abdomen. He was convinced that after all this time he knew the exact condition of every part of his intestine. He said that his transverse colon was in his pelvis, as he could feel it there himself; the beginning of his descending colon

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was, he believed, normal, but it ended in a sort of funnel, the rest of the descending colon being exceedingly small, with a lumen no broader than a lead pencil. He explained the fact that he excreted normal fæces by a comparison with the moulds that earthworms throw up: thus a thin strand of fæces was squeezed through the stricture and then coiled round in the normally-sized rectum; as soon as a moderately large mass was formed he excreted it. In consequence of this extraordinary condition which he thought he had, he dieted himself in such a way that the food should leave only a minimal residue; consequently there was very little to stimulate peristalsis. He wanted Mr. Lane to stimulus necessary for his intestine, which had been inactive for so long a period. The result was that his bowels were regular during the short period he remained under observation. The next series of tracings (Fig. 6) is from a case of tabes. Here, again, the delay is only in the peripheral part of the colon, the progress of the food as far as the middle of the transverse colon being normal. In tabes there is often a deficient nervous supply to the bladder, so that difficulty in micturition occurs. The frequent occurrence of constipation is similarly due to deficient nervous supply to the bowel. The constipation can be easily cured by ordinary purgatives (Fig. 7). By this means it is

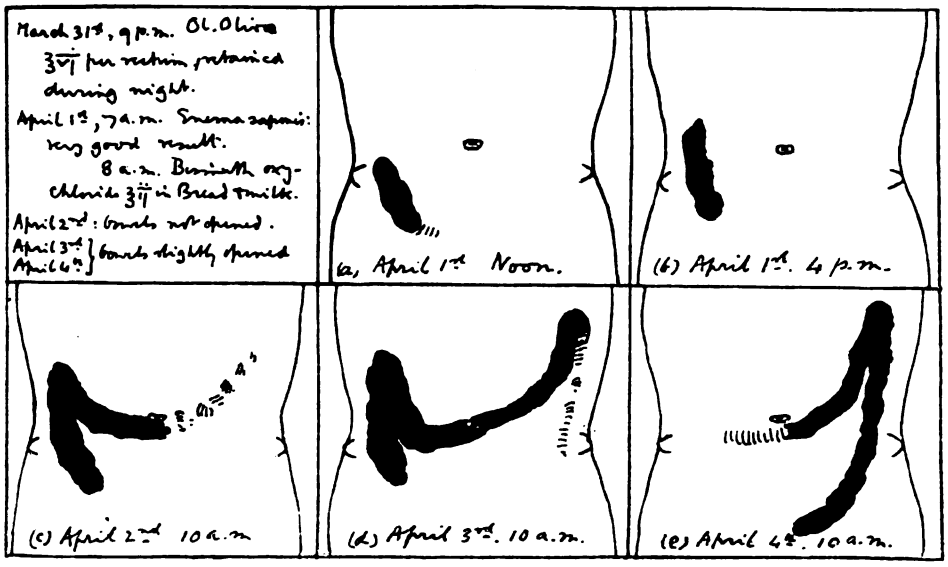
March 31st, 9 p.m. Ol. Olivæ
April 1st, 7 a.m. Enema saponis: very good result.
8 a.m. Bismuth oxychloride
April 2nd: Bowels not opened.
April 3rd
April 4th
Bowels slightly opened
(a) April 1st Noon.
(b) April 1st. 4 p.m.
(c) April 2nd 10 a.m.
(d) April 3rd. 10 a.m.
(e) April 4th. 10 a.m.

FIG. 5.—Constipation associated with hypochondriasis.

remove his colon. Mr. Lane, however, thought that the case would probably turn out to be unsuitable for colectomy, so he asked me to examine him with the X rays. It was found that bismuth reached the cæcum at about the normal time, but that throughout the colon there was a very definite slowing (Fig. 5). But the lumen of the descending colon was perfectly normal, and the only abnormality was that four days instead of one and a half days were required for the series of tracings shown to be taken. I was able to convince the patient that the funnel arrangement which he imagined he had did not exist, and that if he took normal food he would get rid of his constipation. This he did, aperients being also ordered for a time, so as to produce the extra possible to increase the stimulus to the intestinal muscle, the local nervous system of which is still sufficiently active to be able to respond to a strong stimulus.

Now we come to the group of cases (3) in which the constipation is due to insufficient stimulation of peristalsis. Some of these cases are due to too little food being taken, as in the cachexia of tuberculosis and cancer, where the intestinal muscle is also abnormally feeble. In other cases, as in the patient with hypochondriasis, sufficient food is taken to prevent emaciation, but it contains too little indigestible residue. A large number of the milder cases of constipation are due to this cause, and they are all easily cured by a suitable diet.

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Next there are the cases (4) in which persistalsis is inhibited by reflex stimulation of the sympathetic nerves. You know that the intestinal muscles are supplied by motor fibres in the vagi and by inhibitory fibres in the sympathetic nerves. If there is pain in any part of the body, but especially in the abdomen, owing to disease of the stomach, bladder, uterus or ovary, the pain produces reflex inhibition of the intestinal movements, most probably through the sympathetic nerves. The evidence for this is that such cases of constipation can be cured or relieved by means of opium or belladonna; that is to say, instead of giving a purgative to increase the peristalsis, one gives a sedative to diminish the inhibitory action of the sympathetic nerves. Reflex inhibition is also the cause of the complete cessation of gastro-intestinal activity, which follows the perforation of an abdominal viscus, even before the stage of general peritonitis, in which inflammation of the intestinal muscles themselves makes the constipation still more intractable. Then there is the irritative stage of lead poisoning. In lead poisoning two different conditions may give rise to constipation. Firstly, the sympathetic nerves may be irritated by the poison. When that is the case movement along the alimentary canal is unduly slow. If at a later stage the sympathetic nerve becomes partially paralysed (5) instead of being stimulated, the motor impulses have free play uncontrolled by the sympathetic inhibitory action, so that the normal peristalsis, in which contraction behind the fæces and relaxation in front of them causes a steady movement forward of the contents of the canal, is replaced by quite irregular contractions, giving rise to colic in addition to constipation. That such a second stage of lead constipation exists is proved by the fact that a number of cases of lead colic have been operated upon with the idea that they were cases of peritonitis, and coils of intestine have then been seen to be contracted tightly into white cords no thicker than a lead pencil. Fig. 8 is taken from a patient suffering from lead poisoning with constipation without colic. Hence probably there was irritation, and not paralysis of the sympathetic

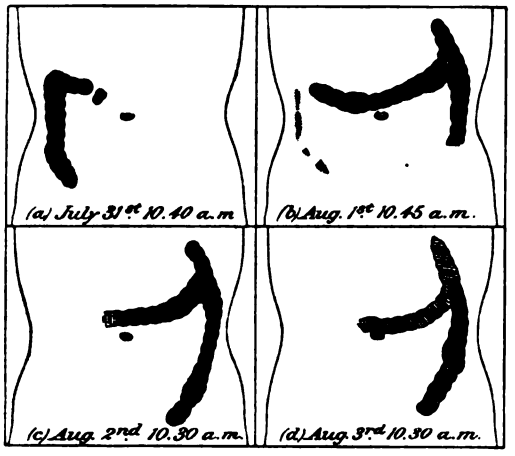


FIG. 6.—Constipation due to tabes dorsalis. Bismuth breakfast at 6.30 a.m. on July 31st.

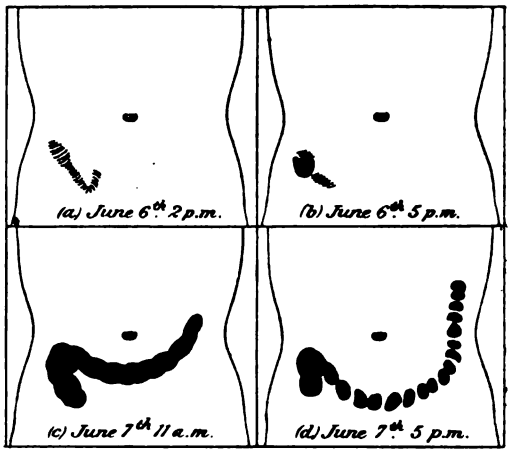


FIG. 8.—Constipation due to lead poisoning. Bismuth breakfast at 6 a.m. on June 6th.

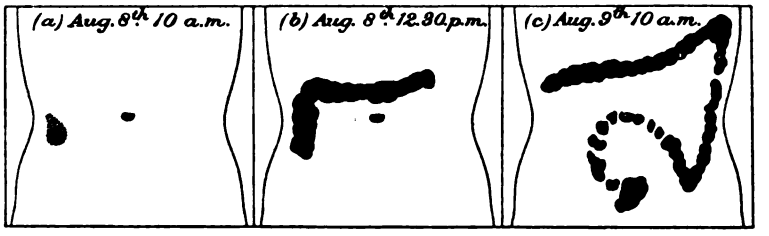


FIG. 7.—Same case as in Fig. 6, under treatment with purgatives. Bismuth breakfast at 6.30 a.m. on August 8th.

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nerve; the irritation of the sympathetic nerve produced a diminution in the activity of peristalsis everywhere, not only in the large intestine but also the small, this being the only case I have seen in which there was definite delay in the passage through the small intestine. At two o'clock, eight hours after food was taken, the bismuth reached a point which it should have attained in four hours. After that, the passage through the rest of the colon was slow, as in the other cases.

We now come to the cases (B) where constipation is caused by obstruction. The first group (1) of such cases consists of those in which the obstruction is due to dry and hard fæces. If in any case of constipation due to one of the causes already considered, or to inability to defæcate, a mass of fæces collects, particularly in the descending colon or sigmoid flexure, it may become so hard and dry that the intestinal muscle is unable to push it forward, so that more or less complete obstruction results.

Another cause of obstruction by dry hard fæces is the drinking of too little water owing to the absence of thirst, or in women owing to their desire to micturate as rarely as possible. In other cases water may be replaced to an abnormal extent by alcohol. The severe constipation of diabetes is partly due to excess of water being excreted in the urine, and that sometimes following hard exercise is due to loss of water by excessive sweating. If the contents of the colon are abnormally hard and dry, the work which the intestinal muscle has to do to push them along is so considerable that a delay in their passage occurs.

In most cases of constipation, in which some obstruction is present owing to the fæces being hard and dry, the condition can be recognised by the discovery of hard fæces in the descending colon or rectum on abdominal palpation or rectal examination. In all such cases it is essential to remove the accumulated fæces before proceeding to any other treatment. This can best be done by enemata. In severe cases it is necessary to soften the fæces by an injection of six ounces of olive oil in the evening; this is retained during the night, and on the following morning an ordinary enema will generally be found to be effective. Frequently the treatment by enemata must be repeated several times before all the retained fæces are removed.

The second group (2) of obstruction cases consists of those in which there is organic obstruction due to diminished lumen of the alimentary canal. It is this condition which one always has to think of in cases of constipation coming on suddenly in people of middle age. It may be due to a growth of the pylorus, when constipation is sometimes the first symptom noticed. Then again there may be a growth in any part of the large intestine. There are rarer cases of congenital and acquired stricture in different parts of the intestine, and occasionally, too, cases occur which are due to obstruction from external pressure, particularly in the pelvis.

The second great group of cases (II) consists of those in which the constipation is due to inability to defæcate efficiently—dyschezia. The majority of cases of very severe constipation, such as those in which the bowels may not be open for several weeks, are examples of this condition, so that ileo-sigmoidostomy, with or without colectomy, which has recently been proposed as treatment for constipation, would clearly be useless, as it would remove a normal colon without curing the inability to defæcate. The inability is due to one of three causes: (A) Loss of the defæcation reflex, (B) atonic dilatation of the sigmoid flexure and rectum, or (C) atony of the voluntary muscles employed in the act of defæcation. The first two of these frequently go together, and they originate in the voluntary disregard of the call to defæcation. This is particularly frequent in girls and women, in whom it is due to false modesty or to laziness. It is sometimes due in both sexes to fear of defæcation on account of the pain incurred in the act, owing to the presence of piles, anal fissure, or other disease in the pelvis. In time a patient, who voluntarily disregards the call to defæcate, allows the fæces to accumulate so frequently and in such large quantities in the rectum and sigmoid flexure that the defæcation reflex becomes impaired. The sensitiveness of the mucous membrane is so blunted that these patients frequently tell you that they have no desire to defæcate, even though you have found by digital examination that the rectum is distended with fæces. The accumulation of fæces distends the rectum and the sigmoid flexure, which consequently become atonic and paretic, so that defæcation is finally impossible. Here is an example of this class of case in a

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girl, æt. 17 years, who was admitted into Guy's for complete constipation of five weeks' duration. Previous to this attack she had been becoming steadily more constipated for seven years. She herself recognised that her constipation was due to her former habit of restraining the desire to defæcate in order to get to school and later on to her work in time, in spite of getting up late. The tracings (Fig. 9) show that the passage through the small intestine and colon was abnormally rapid, but that the fæces accumulated in her enormously dilated sigmoid flexure and rectum. In spite of this she felt no desire to defæcate and she was unable to do so when she made the attempt. By means of enemata, which are the only effective treatment for such cases, the rectum and sigmoid flexure were emptied. For a year now she has used enemata on alternate evenings and has kept well, except for occasional relapses due to neglecting

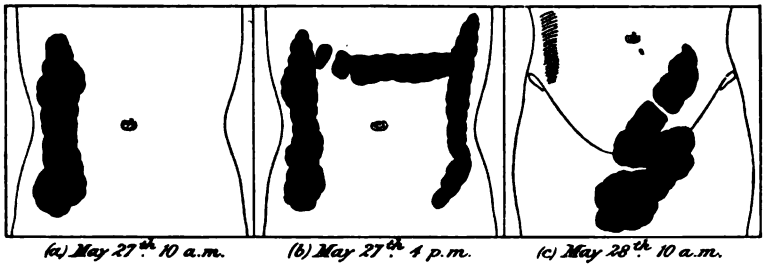


FIG. 9.—Constipation due to inability to defæcate. Bismuth breakfast at 6 a.m. on May 27th.

to use the enemata; on these occasions the skilful administration of an enema by a nurse has always had the desired effect. In some cases of this sort the continual administration of enemata, by keeping the rectum and sigmoid flexure empty, allows their musculature to regain its tone and the sensibility of the mucous membrane to return, so that voluntary defæcation becomes once more possible. Thus a woman, æt. 54 years, had been constipated for thirty-three years, and had been unable to defæcate without enemata for twelve years. She had been taking large quantities of drugs and had used enemata once or twice a week, when it was supposed that the purgatives had caused a sufficient amount of fæces to collect in her rectum for the enema to work upon. The X rays showed, however, that there was very little delay in the colon; the rectum was found to be constantly full, so that the constipation was clearly due to inability to defæcate. For six months the patient gave herself an enema on alternate evenings and attempted each morning to open her bowels without assistance. At last one morning, about two months ago, she found that she could defæcate. Since then her bowels have been open almost daily without the aid of medicine or enemata.

The last class of case (c) is that in which the dyschezia is due to weakness of the voluntary muscles which normally take part in the act. In some women who have borne many children this may be partly due to injury of the muscles of the pelvic floor. But the most important group of cases is that in which the abdominal muscles are weak. This is frequently due to the stretching produced by repeated pregnancies or by the accumulation of fat or fluid within the abdomen. In such cases there is more or less well-marked visceroptosis, and many authorities have thought that the constipation was due to the enteroptosis, the passage of fæces being blocked by kinks at the hepatic and splenic flexures of the colon. But observations with the X rays have shown that these flexures are as sharp in the majority of normal individuals in the erect posture as in the severest cases of enteroptosis. Moreover, I have not yet seen in cases of constipation associated with visceroptosis any delay at the flexures, and in fact the fæces generally reach the sigmoid flexure in normal time. The constipation is due to inefficient defæcation owing to the weakness of the abdominal muscles. By means of exercises the condition of these muscles can be greatly improved, but in the severer cases a good abdominal belt must be used in order to replace to some extent the weak abdominal muscles. The crouching position for defæcation, in which the thighs press on the abdomen, is also of great value in such cases.

I cannot on this occasion fully discuss the question of the symptoms of constipation. But I

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wish to warn you against becoming fascinated by the theory of auto-intoxication, which is so popular at the present day, and which ascribes most of the ills of mankind to "copræmia," or poisoning by substances absorbed from fæces retained too long in the alimentary canal. Personally I doubt whether, so long as no stercoral ulcers have formed, any of the symptoms associated with constipation are due to this cause. Many are simply additional symptoms of the condition, such as neurasthenia or chlorosis, which has given rise to the constipation. This is also the case in visceroptosis, in which a suitable abdominal belt, by preventing the drag of the viscera on the attachments of the mesentery, relieves the pain and other disturbances at once, even before the constipation has disappeared. Some of the symptoms of the slighter forms of constipation, such as headache, a feeling of slackness and slight abdominal discomfort, are due probably to a reflex from the distended rectum, as it is a matter of common experience that they are relieved instantaneously when the bowels are well opened, although this would be impossible were they due to auto-intoxication.

Certain neuralgic pains are probably caused by pressure on nerves, and the frequent association of hæmorrhoids with constipation is often due to local interference with the circulation by the pressure of hard fæces accumulated in the rectum. When there is any considerable degree of obstruction from impacted fæces, flatulence may be troublesome and colicky pain may occur. Defæcation is generally more or less painful, and the straining required may give rise to hernia, prolapse of the rectum or uterus, syncope or cerebral hæmorrhage in those disposed by pre-existing abnormalities to these conditions.

Finally a catarrh of the rectum with spurious diarrhœa and tenesmus may be brought on by the irritation of a fæcal accumulation, and impaction of fæces may occasionally produce stercoral ulcers in the rectum, sigmoid flexure or other parts of the colon. These ulcers on rare occasions perforate and produce local or general peritonitis, and they have been known to produce intestinal obstruction by their subsequent cicatrisation.

## The Causes of Constipation.

### (I) Slow Passage through the Intestine.

A. *Deficient motor activity.*

(1) *Inefficient intestinal muscles.*

(*a*) "Constitutional."

(*b*) Senile.

(*c*) Anæmia, especially chlorosis.

(*d*) Cachexia of tuberculosis and cancer.

(2) *Depression of central and peripheral nervous system, resulting in diminution in the response to stimuli which normally produce peristalsis.*

(*a*) *Functional.*

(i) Neurasthenia, hypochondriasis, melancholia.

(ii) Old age.

(*b*) *Organic.*

Tabes.

(3) *Insufficient stimulation of peristalsis.*

(*a*) Too little food.

(*b*) Food containing too little indigestible residue or chemical stimulants.

(4) *Stimulation of sympathetic nerves inhibiting peristalsis.*

(*a*) Reflex from painful organ.

(*b*) Irritative stage of lead poisoning.

(*c*) Peritonitis.

(5) *Paralysis of sympathetic inhibitory nerves permitting irregular peristalsis.*

Paralytic stage of lead poisoning.

B. *Obstruction.*

(1) *Dry, hard fæces.*

(*a*) Resulting from fæcal retention, brought about by any of the other causes of constipation.

(*b*) Too little water.

(i) No thirst.

(ii) In women to diminish frequency of micturition.

(iii) Replaced by alcohol.

(*c*) Excessive secretion of urine—diabetes.

(*d*) Excessive secretion of sweat—after hard exercise and in hot weather.

(2) *Diminished lumen of alimentary canal.*

(*a*) Congenital or acquired stricture, including growth: œsophagus, pylorus, intestine, anal.

(*b*) Pressure from outside.

(*c*) ? Kink.

### (II) Inability to Defæcate (Dyschezia).

A. *Loss of defæcation reflex.*

B. *Atonic dilated rectum and sigmoid flexure.*

A and B generally together due to habitual disregard of call to defæcation, owing to sloth, modesty or fear of pain from piles, fissure or other pelvic disease.

C. *Atony or paralysis of voluntary muscles concerned in defæcation.*

(1) *Abdominal muscles.*

(*a*) Organic paralysis.

(*b*) Weak from repeated pregnancies, etc., associated with visceroptosis.

(2) *Pelvic floor muscles,* which are liable to be injured during parturition.

(3) *Diaphragm.*

*August 3rd,* 1908.

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# THE CLINICAL JOURNAL,

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## A CLINICAL LECTURE

ON

## A CASE OF HOMONYMOUS HEMIANOPIA WITH UNUSUAL SENSORY SYMPTOMS.

Delivered at St. Bartholomew's Hospital.

By H. H. TOOTH, C.M.G., M.D., F.R.C.P., Physician to the Hospital.

GENTLEMEN,—The following case was shown to my colleagues at medical consultations in this hospital, and I thought it would be a good opportunity, as the patient is still in the hospital, to say a few words on the very interesting subject of hemianopia in general.

The patient, a veterinary surgeon's assistant, æt. 41 years, was admitted to St. Bartholomew's Hospital with the following history. On February 8th, at 7 p.m., he found that his right arm and leg and the right side of the body became suddenly cold. The arm felt numb from the wrist to the elbow, with a tingling sensation. There was some tremor and want of extensor power in the thumb and fingers. He also found that he could not use the clippers in clipping his dogs as easily as formerly, and that there was some diminution of the right field of vision. The toes of the right foot were "drawn up" but he had no difficulty in walking. He thinks his memory also was slightly impaired. On February 11th he had some occasional deafness of the right ear with giddiness. On February 12th the right half of the field of vision became absolutely blind and has remained so.

There was a history of syphilis fifteen years ago with no tertiary symptoms. From that time, however, he completely lost his sense of smell. Five and a half years ago he fell and fractured the "right side of the skull," also the nasal bones with a resulting depression of the bridge. On admission the patient gave a good account of himself, and

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was of average intelligence. There was no facial asymmetry, he could close the eyes independently and his tongue was not deviated but very tremulous. There was complete right homonymous hemianopia and a perimeter chart showed some diminution of the left visual fields. The right pupil was larger than left. Both acted to light equally well on either half of the field. Mr. Spicer's report on the condition of the discs on February 26th was: "The discs have good colour: there are inequalities in the bore of the veins, and the arteries are perhaps small." There were languid nystagmic movements on conjugate deviation of eyes to left. There was slight weakness of left external rectus. Hearing was obtuse in both ears, more noticeable on the right side. There was complete anosmia. Examination of the limbs of the upper extremity showed that his grasps were fairly powerful, but the right was distinctly less so than the left. Fine tremor of right hand was noticed but it was not constant. Examination of the lower extremity showed that his gait was natural, but he was a little unsteady standing with his eyes shut. The knee-jerks were rather exaggerated. There was ankle clonus on the right side. Tactile sensation tested by cotton-wool and light touches seemed to be perfect over the right half of the body, but stimuli were not quite accurately localised and the error was inconstant; there was some slight delay in recording stimuli on the right ride below the clavicular level, when compared with the left. Sensation to pain seemed to be quite natural. *Temperature* sense was distinctly altered over the right half of the trunk below the clavicular level, and in the right arm and leg. He could appreciate cold distinctly, but not heat with any certainty. When a cold test tube was applied two or three times he felt it correctly as cold, but almost invariably a hot test tube was felt as cold also.

The first point in this case which calls for consideration is the onset, which was gradual. On February 8th he experienced a sensation of cold, numbness, and tingling, mostly in the right arm. And I daresay from that time dates the thermo-anæsthesia, which is so unusual a feature in the case. These symptoms suggest that he must have had some lesion, of what nature we can only conjecture, of that part of the internal capsule which receives the sensory fibres from the body by way of the fillet, namely, its posterior part, and perhaps the optic thalamus. There was also some weakness of the right arm and hand, which still persists slightly, indicating that there must have been some affection also of the motor fibres of the capsule. Four days afterwards a very definite symptom appeared, namely, hemianopia. I shall speak of that more fully presently. This gradual onset must either indicate that there has been a thrombotic lesion of vessels—and of what vessels I shall probably indicate later if I have time—or there is a rapidly growing tumour. This latter is not very likely. At any rate it cannot be a tumour which has attained any great size, because at present there is no optic neuritis. I should think it is more probable that this is some vascular lesion. There is a past history of syphilis, and there may be a small gumma pressing on vessels, but it is more likely that there is a thrombosis secondary to specific endarteritis.

The next point I want to consider in connection with this case is the order of the symptoms: sensory first, that is to say, coldness and numbness; motor next, namely weakness of the right hand; and then at a considerable period afterwards, loss of sight on the right side of the visual field. Last of all there are the residual sensory symptoms which have remained up to the present time, that is to say, the alteration in the appreciation of heat and cold.

Now to consider one or two points more in detail. The most interesting feature in connection with this case is the loss of sight on the right side of the field of vision due to blindness of the left half of the retina. May I draw your attention to this ordinary text-book diagram which is given to explain the peculiarities of the fields of vision. Let us trace the fibres. Fibres arising from the left half of both retinæ pass along the optic nerves to the optic chiasma, and on in the left optic tract to the left corpora geniculata, forming connection with the optic thalamus probably. So that the fibres of the left eye keep on the left side, while those of the left or internal half of the right eye cross in the chiasma. The fibres take a fresh start, as it were, from the corpora geniculata and go on as a well-defined mass of fibres in the posterior lobe of the brain, known as the optic radiations. Those radiations finally dispose themselves in the cortex of the posterior lobe, principally in the region of

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the cuneus, but probably also entering into connection with the angular gyrus, so that we may divide the visual apparatus and path for convenience into five sections: (1) the optic nerve, (2) the optic chiasma, (3) the optic tract (from chiasma to corpora geniculata inclusive), (4) the optic radiations, (5) the cortex of the posterior lobe, the cuneus. Let us for a moment touch on the effects of lesions in particular situations in the course of these optic paths. It is scarcely necessary to say that a complete lesion of the optic nerve causes blindness of the same eye. It is also quite obvious that a similar lesion of the optic chiasma will result in blindness of both eyes. I pass round a photograph of a tumour of the chiasma, for instance, in which the result was complete blindness. Partial lesions of the chiasma occasionally occur. For instance, a small local lesion of the middle part of the optic chiasma would give rise to temporal hemianopia, because it would cut off the fibres to the nasal side of the fundus on both sides—a rare condition. Still more rare is a lesion of both sides of the chiasma producing "heteronymous" nasal hemianopia. Lesions of the rest of the optic path are common and our powers of diagnosis fairly adequate.

A lesion of the left optic tract, say, will give rise to right homonymous hemianopia, because it will produce blindness of the left (temporal) half of the left eye, and of the left (nasal) half of the fundus of the right eye. But the same disorder of vision will occur in lesions of the optic radiations and also of the cuneus. We have, however, a means of diagnosing whether the lesion is in this section of the system the optic tract, based on the fact that this tract is concerned in the reflex contraction of the pupil to light. A beam of light thrown on the blind half of the fundus causes no contraction of the pupil in optic tract lesions, whereas if the lesion is posterior to the corpora geniculata the pupil contracts as readily to light stimuli on the blind as on the normal half of the fundus. This is known as Wernicke's sign. In our patient this sign is not present, and we may assume therefore that the lesion is further back than the optic tract. A lesion involving the optic radiations, such as a tumour or softening, will again produce hemianopia. I pass round the brain of a man who had an abscess in the left thalamic region, evidently causing pressure on the optic radiations, together with softening involving the thalamus, and the motor and sensory fibres of the capsule. He had hemiplegia, hemianæsthesia, and hemianopia on the same side. The localisation was fairly complete, and an operation was performed by Mr. Ballance in order to evacuate the abscess, which, however, was encapsuled, and the pus was so thick that no relief followed. The patient died. He had word-blindness, due no doubt to inflammatory involvement of the angular gyrus. I pass round some photographs of this case. I show you also the brain of a man, æt. 65 years, who came into the hospital with a number of indefinite symptoms of a mental character, rather suggesting that he was the subject of general paralysis of the insane, in fact he became maniacal and had to be removed to Bethlem, where he died. The only paralytic symptoms during life were slight weakness of the right arm, with paralysis and rigidity of the right leg; no anæsthesia nor hemianopia. The brain shows a large area of softening, no doubt due to thrombus of the left posterior cerebral artery, situate in the middle of the posterior lobe, but extending forwards to involve slightly the posterior part of the optic thalamus. This case serves as a sort of negative to the last, and shows how large a lesion may exist in the posterior lobe and even in the optic thalamus without eye or sensory symptoms. The paralysis of the leg must be due to involvement of the leg fibres in the corona radiata, but the case also shows what serious mental symptoms may be associated with lesions of the posterior lobe.

Now a few words in discussion of the sensory side of our case. All the fibres of the spinal cord that have to do with sensation must enter at the posterior roots, and after entering pass up the cord in the posterior columns, finally ending in the medulla in the nuclei of the funiculus gracilis and the funiculus cuneatus. From these nuclei the fibres take a fresh spring and run up as the fillet. The fillet fibres finally gather themselves up, to end as Horsley suggests in the optic thalamus. Possibly a certain number do not go to the optic thalamus, but in the posterior part of the internal capsule. Their further destination is at present conjectural. Lesions of the internal capsule are often associated with loss of sensation; in fact it is impossible to have a hæmorrhage of any size in the internal capsule region without accompanying loss of sensation on the opposite side for a time. Lesions

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of the optic thalamus, such as tumours, are also associated with alterations of sensation on the opposite side, due either to affection of the fibres which enter the optic thalamus or some central mechanism there. The fillet fibres decussate, and very nearly at the same point in the medulla oblongata as the motor fibres do, namely, at the post-pyramidal decussation, so that the resulting hemianæthesia is, with the motor paralysis, on the opposite side to the lesion. The form of anæsthesia is usually tactile, rarely pain or temperature. And a very common form of tactile loss of sensation is erroneous localisation, or atopognosis. You ask a patient to point to the spot which you touch, and he will point to a place some distance away, from the point stimulated, but the acuteness of sensation seems to be unaltered. This error may be *inconstant*, that is, at a point proximal, distal, or to one side of the point touched, a form of atopognosis common in all forms of altered sensation, peripheral, cerebral, or functional. But in atopognosis of strictly cortical origin the error is frequently *constant*, that is regularly felt at a point proximal to the spot stimulated, and at about the same distance, which is also constant for the same case, though very variable in different cases.

The dissociation of temperature sense found in our case is a very unusual symptom in cerebral lesions in my experience. This symptom is generally associated with central spinal lesions, of which syringomyelia may be taken as the type.

Now, supposing this to be a vascular thrombotic lesion in our case, what vessels are occluded? A neat diagnosis of occlusion of a single vessel offers some difficulty. The whole subject of the distribution of cerebral vessels has been revised lately by my colleague, Dr. Beevor, in an admirable paper before the Royal Society, in which were embodied the results of injections of the several systems of vessels with coloured fluids, thus mapping out their areas of distribution in colours. In a horizontal section of the brain you see these well-known objects: the nucleus caudatus, the optic thalamus, the nucleus lenticularis, and the V-shaped internal capsule with its anterior limb, its genu, its middle and posterior limbs.

It would be impossible in the short time at my disposal to review this subject here, but I wish to draw your attention to the blood supply of the internal capsule, which is much more complicated than was thought to be formerly. The middle cerebral in passing over the locus perforatus anticus sends in a number of little vessels at right angles to its axis; these supply the nucleus lenticularis and the central parts of the corona radiata, only a small part of the capsule. Nevertheless it is these vessels that so often are affected by atheroma, and the rupture of which cause hæmorrhages, pressing mechanically upon or destroying the fibres of the capsule with resulting hemiplegia.

The blood supply of the internal capsule is as follows: The anterior limb or anterior third is supplied by the anterior cerebral. The upper part of the genu of the capsule is supplied by the middle cerebral, a comparatively small part of the whole capsule. The internal part of the genu receives its blood from the posterior communicating artery. The posterior limb, or third, is nourished by the anterior choroid, which comes off from the internal carotid close after the posterior communication. This vessel also supplies part of the optic radiation, and its occlusion may therefore cause hemianopia with hemianæsthesia, and possibly some hemiplegia. If therefore our case is one of vascular thrombosis, occlusion of the anterior choroid may perhaps best explain the clinical features. This important function of the anterior choroid artery was first pointed out by Kolisko in 1891, but Beevor's work has not only confirmed this observation, but has also completely mapped out the blood supply of the capsular region.

The optic thalamus, most of the posterior lobe of the brain, together with a great part of the corona radiata, including the optic radiations, are supplied by the posterior cerebral. So that among other things occlusion of the posterior cerebral would cause hemianopia and hemianæsthesia. A thrombotic lesion of the posterior cerebral might explain therefore the symptoms in our case, but perhaps not so satisfactorily as a similar lesion of the anterior choroid as suggested above.

*August 10th*, 1908.

**Corrosive Sublimate Applied without Pain.**—Appleman (*American Medicine*) states that when solutions of mercuric bichloride are made with normal salt solution instead of water, they do not cause the slightest pain when applied to the mucous membranes. — *Med. Record*, vol. lxxiv, No. 3.

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# RHEUMATISM.

A Discussion at the Hunterian Society.

**Mr. ROWLAND HUMPHREYS in the Chair.**

Dr. Fredk. Taylor. Dr. Otto Grünbaum.
Dr. F. J. Poynton. Dr. Seymour Taylor.
Dr. Horder. Dr. George Oliver.
Dr. Luff. Dr. Fortescue Fox.
Dr. Fred. J. Smith. Dr. Leonard Guthrie.
Dr. Arthur T. Davies.

---

Dr. Taylor, in opening the discussion, said: When, sir, I was honoured with the request from your Council that I would open a discussion on "rheumatism" at this Society it was intimated to me that I had *carte blanche* with regard to my treatment of it. And having agreed to undertake the task, I thought I might avail myself to the full of the privilege of one who opens a debate, that is, to spare myself the presentation of any new matter, whether facts or theories, with regard to any one branch of what is really a very large subject, and to indulge in some general considerations and in numerous inquiries, few of which I shall be able myself to answer, and some of which even my audience may not be in a position to respond to.

I am afraid that I may be looked upon as being in the position of the trumpeter in the old story, who was reproached with exciting other people to battle, while he himself escaped all danger. I am not sure that the buglers of the present day are quite so fortunate in respect of their safety, or so contemptible in regard to their honour. I do not suppose I shall do much damage with what weapons I am wielding, but I daresay I shall not escape the shots or bullets of some of the combatants.

Rheumatism is a very comprehensive term, and if one were to take it in its widest sense it would indeed be a task beyond the limits of an evening's discussion, and certainly beyond the limits of the time which one who introduces it could devote to it.

I will say at once that I do not consider it desirable, so far as I am concerned, to do otherwise than take the word in its most restricted sense—that is, rheumatic fever or acute rheumatism—and to discard for the present all such conditions as have received the epithet rheumatic, either because they are supposed to have arisen from cold, or because they are painful affections, or because they are articular inflammations of obscure or doubtful origin.

What we surely have to consider is the pathology, diagnosis, and treatment of the disease known as "acute articular rheumatism," and to work out its relations to such other pathological conditions, cardiac inflammations, cutaneous eruptions, or other lesions, as may be supposed to be caused by it, or to have an origin in common with it.

At the bottom of the whole subject lies the question of the intimate pathology of acute rheumatism, and I take it that no real progress can be made until we are all agreed as to the exact pathology of this typical disorder.

No one can realise this, perhaps, better than those who remember the position with regard to tubercle some thirty-five years ago. In the year 1873 the Pathological Society was engaged on three separate evenings in a discussion on the anatomical relations of pulmonary phthisis to tubercle of the lung. Every conceivable theory with regard to the relations of grey tubercle, of yellow tubercles, pneumonia, fibrous tissue, calcareous deposit, etc., was discussed; and the possibility of the inoculation of tubercle was recognised, but little of a conclusive kind was arrived at. At length, a few years later, in 1882, the whole mystery in all its multiple phases was made clear by Koch's discovery of the tubercle bacillus.

Rheumatism has been explained by numerous theories, humoral, chemical, nervous; all pushed strongly and ably by their advocates, but none accepted by us all. Gradually the close resemblance of the disease to certain other infective diseases, such as pyæmia and gonorrhœal synovitis, forced itself upon the mind, and the conviction grew that it must be due to an infection not materially different, whether as toxin or organism, from what was present in those disorders.

I understand that we shall have here this evening those who have satisfied themselves that they have discovered the organism of rheumatic fever, and so strongly do I feel that this is the key of the position that I believe it is futile for one like myself, not a bacteriologist, and knowing what I do know in this respect only at second hand, to attempt to discuss the pathology of the disease from the bacteriological standpoint.

Personally, I am convinced that it is an infectious disorder, in all human probability, one

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would almost say with certainty, due to a micro-organism, and I wait with the greatest interest what Dr. Poynton has further to add to the valuable communications he has already made on this branch of the subject. I have only this to remark, that the history of bacteriology shows us that the most rigid proof must be demanded as to the pathogenicity of any organism that may be found in a given disease; and the profession is generally justified in withholding its assent to the recognition of an organism as the cause of a disease until there is a considerable consensus of opinion amongst a large number of observers that such organisms can be, and are constantly, found by skilled bacteriologists.

I turn now to the relations of rheumatism to other disorders once thought to be entirely distinct, and the first that commands our attention is chorea.

Here we have a complaint of which the clinical associations, equally with its pathological appearances, demonstrate to be of infective or toxic nature rather than a purely nervous disease; or rather, to be a disease in which the failure of function of the parts concerned is clearly brought about by a toxin, and is not determined solely by such emotional or intellectual disturbance as fright, or irritation, or overwork.

The facts which apart from any bacteriological study must convince all of us that the disease belongs to the infectious group are—(1) the occasional occurrence of endocarditis in cases in which there has been no previous rheumatism; (2) the great frequency with which endocarditis is found to occur in fatal cases; and (3) the striking resemblance which holds between the modes of death in some fatal cases of chorea and in two other nervous diseases, in which—bacteriology or no bacteriology—infection cannot be doubted; I mean hydrophobia and tetanus.

Indeed, these three diseases are curiously alike and different. Alike now in being almost exclusively infective disorders of the central nervous system; different in the area of that nervous system chiefly affected; different in the virulence of the poisons and hence in the fatality of the disease. For chorea is only exceptionally dangerous to life, hydrophobia is inevitably fatal when the latent period is passed, while tetanus holds an intermediate position.

But what I want to call attention to especially in this connection is that in many deaths from these diseases, involving, as each of them does, a great expenditure of muscular force in involuntary contractions or spasms, the patient does not die right out from exhaustion but may present a period of quietude for some hours before death, which may give the observers a hope of recovery never to be realised. I have seen this markedly in cases of acute chorea, as well as in tetanus; and I have seen it in hydrophobia, where a patient after a period of violent excitement and faucial spasm drank freely a quantity of beef tea, but nevertheless died a few hours later, the total period of his illness being no longer than what is usually observed in this fatal disease. What share muscle-poisons may have in this final condition I am not in a position to say, but at present I incline to believe in the persisting influence of the original virus, whether choreo-rheumatic, or rabic, or tetanic as the case may be.

Chorea, then, is no doubt toxic, in spite of the frequent assertions, not always to be explained, that the symptoms have first appeared after fright, or other emotional or mental strain.

Again, it remains with the bacteriologist to provide us with the sufficiently convincing evidence that the toxæmia is due to a micro-organism; that the micro-organism is the same as that which is the cause of rheumatic fever.

It is not enough to point to the frequency with which chorea occurs in patients who have had rheumatic fever; with which rheumatism occurs in a patient who has had chorea; with which choreic movements occur in the actual course of a rheumatic attack; with which pregnant females who have a severe or fatal chorea have previously had rheumatism. Some physicians abroad have not, I believe, committed themselves so unreservedly to the exclusively rheumatic origin of chorea, but believe that other infectious disorders, such as measles or scarlatina, may be sometimes the antecedents of chorea.

It must be admitted that this position is much more difficult to support by bacteriological evidence, seeing that we still seem to be a long way from a recognition of a specific organism in either of these exanthems.

Nevertheless, it is essential at the present day to remember that any structural and functional

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deviation from the normal is not necessarily due to one specific cause alone. To take synovitis itself, there are, we know now, several different infections which may produce forms of synovitis, so much alike that it is almost impossible to distinguish them from one another, except with the aid of the history or the associations of the case. The same may be said of the throat, where, for instance, diphtheria, follicular tonsillitis and Vincent's angina may have almost identical appearances, requiring the help of the bacteriologist to discriminate between them.

It is not therefore unreasonable for our foreign *confrères* to believe in the possibility of a scarlatinogenous, or morbilligenous chorea, though I am quite prepared to believe that the bacteriological evidence will now at least be all in favour of the rheumatic connection alone.

When we proceed to the other *complications* of rheumatic fever or *manifestations* of rheumatic fever the difficulties increase. I refer to tonsillitis, to erythemata of different kinds, especially erythema nodosum, to purpuric eruptions, and peliosis rheumatica.

Even before rheumatism was held to be a microbic affection, its frequent association with these local lesions was well known, and conversely it was known that skin diseases of the erythematous order were frequently accompanied by joint pains or joint effusions whose connection with acute rheumatism was not proven or even sought to be proved. As an instance I may mention erythema exudativum multiforme.

The purpuric eruption which occurs sometimes on the legs of patients with acute rheumatism, and is distinguished by its almost scarlet colour and closely-set small maculæ, is sometimes called Schönlein's disease, or purpura rheumatica; and in this simple form—seeing that it comes with a definite attack of acute rheumatism and disappears with subsidence of the pyrexia and arthritis—can not be regarded as otherwise than rheumatic in origin. But the name Schönlein's disease has been extended, by Professor Osler at least, to any form of purpura, erythema, or even urticaria, occurring in association with joint pains or joint inflammations.

It is surely out of the question for us to regard all these cases as rheumatic, in the restricted sense of the term.

The work of recent years has demonstrated that there are many forms of infective arthritis besides acute rheumatism; and it is as reasonable to suppose that these mild joint affections in association with erythema and urticaria are due to some other condition—some other toxæmia probably—as to suppose that they have a bacteriological alliance with rheumatic fever. Possibly it may be shown that their inclusion in one group, rather than in two or three, is not justifiable.

According to Sir Almroth Wright the hæmorrhagic, erythematous, and urticarious lesions are often the result of a deficiency of calcium salts in the blood; and even if this were admitted to be universally true, the link between this hæmic deficiency and the sometimes obvious cause of the disorder has to be worked out. Should, however, this be an essential feature in the pathology of the erythemato-urticarious group, I fail to see at present that it brings that group especially within the range of acute rheumatism.

Indeed, from the point of view of acute rheumatism and the rheumatic poison, I am myself a long way from taking the position that an erythema, whether erythema nodosum or erythema multiforme, is necessarily a *manifestation* of rheumatism, or in plain English rheumatism of the skin.

For we must be as clear and definite in our conceptions as possible. The expressions, manifestations of rheumatism and evidence of rheumatism, must not be used heedlessly. If in a person who has never previously had rheumatic arthritis or rheumatic carditis, or any other ailment even remotely allowed to be rheumatic, an attack of erythema nodosum occurs, then he must have at that time, to justify a belief in the rheumatic nature of the node, acquired the organisms of rheumatism, and must be harbouring them in his economy. Until that has been shown by ocular demonstration, or until it has been shown that erythema nodosum occurs only in those who have had previous unmistakable rheumatism, it can scarcely be asserted that it is essentially and necessarily a rheumatic complaint.

With regard to tonsillitis it seems to me even more difficult to look upon it, whether in its suppurative or follicular form, as necessarily due to rheumatism, in cases where no syphilis, scarlatina or other exanthem is present. It is

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sufficient to admit that the tonsils are especially vulnerable to different disease toxins or their causative microbes, and that rheumatism is one of the many diseases which may supply such toxins.

It will be seen that I have a mind open to conviction in regard to bacteria, and that I should welcome the demonstration of such organisms in regard to all the lesions associated with rheumatic fever, when their existence can be safely and certainly alleged. I think there is nothing more striking in this respect than the discovery of the syphilitic organism, the *Treponema pallidum* and *Spirochæta pallida*, which has now been found in so many local lesions in that disease.

One very interesting question with regard to rheumatism must, I believe, also wait for its solution the skill of the bacteriologist, and that is the relation of acute rheumatism to the diseases called chronic rheumatism and osteo-arthritis. Short of that, it is very doubtful if any convincing evidence could be brought forward to show that there was an invariable connection between acute rheumatism on the one hand and either of these diseases on the other.

Osteo-arthritis of middle-aged and old people is very widespread, and of course occurs in thousands who have never had acute rheumatism.

Osteo-arthritis in its acute form, whether rightly so-called, or more correctly acute rheumatoid arthritis (as some would have it) occurs in just the same young people as are liable to acute rheumatism, and the clinical differences make it extremely unlikely that it can be due to the same virus or organism. And although it is conceivable (for few sciences are more full of surprises than what one may call pathological biology, as witness the newer developments with regard to trypanosoma, Leishman-Donovan bodies, and the spirilla of syphilis and relapsing fever) that all those idiopathic infective, yet not contagious, diseases of the joints may be due to one organism, we can scarcely regard it as probable in the present state of pathological knowledge. And while, since I have limited myself to acute rheumatism, I am not going to enter into a discussion of these arthropathies, which I believe are not "rheumatic" in the sense I am employing the word, I may be allowed to point out that they are among those numerous disorders which have been referred to *autotoxins*, intestinal or otherwise, and that this field of autotoxins has not only not been fully cultivated, but it has scarcely yet been ploughed.

When dealing with the complications of rheumatic fever, it may seem not so much wanting in completeness as to be ignoring the Prince of Denmark in the play of "Hamlet," if I say nothing about endocarditis and its allied conditions, myocarditis and pericarditis. But I have little to say about them that is not already known; and few difficulties to raise as concern the acuter developments, while to touch upon the chronic changes would carry one into a subject—that is heart disease—which is not part of the matter before us, and would require far more time for its discussion than rheumatism itself. Two points alone in this connection will I refer to as requiring some explanation. One may be cleared up by our further bacteriological acquaintance with the disease, and that is, that endocarditis is so constantly limited to the valves, unless as in the case of malignant endocarditis the organisms, which are so obvious then, are conveyed by direct contact or by gradual extension from the first injured valves to other parts. The fact that the valves are constantly moving, or subject to friction of the blood, does not seem to me to be an adequate explanation of their almost exclusive liability.

Another well-known clinical experience of endocarditis is that the first evidence of its occurrence, derived from auscultation, is a systolic murmur, either at the aortic orifice or at the mitral orifice, and rarely a diastolic murmur. Thus the first change in the valves produced by the inflammation, whether a few vegetations or a mere swelling of the valve, produces mechanical results of an obstructive nature in one valve—the aortic; whereas in the other valve—the mitral—no obstructive signs are generally obvious, but the morbid sound is suggestive of incompetence or regurgitation.

There is one complication of rheumatism which is a profoundly interesting mystery, the solution of which we may expect only when the other bacteriological problems have been decidedly settled. I refer to hyperpyrexia. Claimed as a convincing argument by the advocates of a nervous theory of rheumatism, it is equally valuable as a confirmation of the modern toxæmic views. Even now we can scarcely quarrel with the term "cerebral rheumatism," which was given to it before the use of the clinical thermometer, so long as that name does not

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carry with it the idea of a tangible encephalitis or meningitis; but it was with the introduction of the thermometer that a true explanation of its accompanying symptoms was possible.

What I wish to allude to here is the historical fact that a generation ago, that is from the years 1873–1877, there was in the British Isles what one may call an epidemic of these hyperpyrexial cases, which, as far as I know, has not been repeated—at least in metropolitan practice. A committee of the Clinical Society reported upon a number of these cases, without arriving at any satisfactory conclusion as to their origin, and without discovering any factor or occurrence in the history or condition of those who suffered from this complication different from what happened in other more fortunate patients. The cases are now happily infrequent; if promptly treated they can generally be saved, and I do not remember to have read of any case having been examined by modern methods post-mortem, so as to further our knowof their nature. A time may again come when they will be frequent, when the unknown factor which was in force in the seventies may again exert its influence. It will occur to many that our success with the salicylate treatment is a reason for this immunity from hyperpyrexia in rheumatism. But as a fact the salicylate treatment was introduced, though perhaps not fully pushed, during those many years in which hyperpyrexia was rife; and, on the other hand, there was a relative immunity before that time in the years when not the salicylate treatment, but the alkaline treatment was in vogue. The present immunity from this complication is illustrated by the records of Guy's Hospital medical wards, where during eighteen years (1888–1905 inclusive) the admissions for acute rheumatism were 1887, and the cases of hyperpyrexia were only seven. Of these two were fatal, but I am not in a position to state the degree of severity of the remaining five.

The treatment of every disease should have the first and not the last claim on our attention, as it is the professed object of the great majority of medical men. And I remember very well when, some dozen years ago, I read a paper, or rather introduced a discussion, on the subject of anæmia in the Section of Medicine, at an annual meeting of the British Medical Association, and had raised all possible questions as to the pathology and ætiology of anæmia in its different forms, and was hoping to hear something new on the genesis of those obscure forms of the disease, chlorosis and pernicious anæmia, that the very first speaker, and a distinguished speaker, swept aside the whole subject of pathology and ætiology, and proceeded to consider the different preparations of iron which were most efficient in chlorosis. Now I am far from wishing to exalt the subject of pathology beyond what it can rightfully claim, nor would belittle the importance of treatment in any disease whatever. Still, at the bottom of efforts to remove disease, whether by prevention or cure, is the necessity of knowing the causes, and that justifies the time I have already devoted to that part of the subject.

Acute rheumatism at present seems to be, so far as the acute articular manifestations of the disease are concerned, somewhat in the position of syphilis and malaria, that is, a disease for which a more or less satisfactory specific remedy has been found—a remedy which, in each case, has been successfully employed long before anything was really known as to the nature of the disease.

Salicin and the salicylates were introduced some thirty to thirty-five years ago and have been continuously used ever since. Immediately prior to this the alkaline treatment was almost universally adopted in England, combined with the use of opium when the pains were very severe. Dr. Fuller was the great advocate of this treatment, which it was admitted could not be relied upon in all cases. Dr. Fuller insisted that a healthy state of the *primæ viæ* was a *sine quâ non* for successful results; and that so long as the tongue was furred the alkaline treatment was not having a fair chance. Now, as the characteristic of rheumatism is a white thickly-furred tongue, it will be seen that if in any case the alkaline treatment did not come up to expectations, an explanation of this failure and an excuse for its inefficiency could almost always be found.

Sceptics were not wanting, and Drs. Gull and Sutton in the wards of Guy's Hospital treated numbers of cases with no drug other than water flavoured with mint. They claimed that the disease recovered as well as it did under the alkalies—at any rate, that it often got well of itself. Dr. Southey also insisted that rheumatism was a disease that ran its course independent of treatment, and was exceedingly liable to relapse.

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There can, however, be no doubt that the salicylates have given much more satisfactory results. For a time these were given in inadequate doses, and relapse of the pain and fever was common; for a time after this they were often given in unnecessarily large doses, for instance 20 grains every two hours of sodium salicylate. This eased the pains promptly, but it sometimes caused unpleasant symptoms of salicylism, such as headache, deafness, singing in the ears, sometimes delirium, and occasionally epistaxis.

The dose of 20 grains every four hours seems to have done all that was possible by this means of treatment, and will probably continue to be the authorised dose in all ordinary cases.

There remains, however, the question of how to prevent carditis in its different forms, and until that has been satisfactorily solved the profession must still feel that it has not accomplished all that is desirable in regard to the treatment of this disease.

If it should have to be admitted that rheumatic patients are just as liable to develop endocarditis as they did forty years ago we should have to confess that very much remained to be done, and that we could not sit down satisfied with our results. The question of the influence of salicylic treatment upon the occurrence of carditis is a difficult one to answer, and perhaps it can only be answered by collecting reports of a very large number of cases and treating them statistically—a labour which is not always a labour of love, and often leaves one after a year or two's work with very little in exchange. One would, nevertheless, be interested to hear from anyone joining in the debate this evening what evidence can be brought forward bearing upon this point; and I should be too glad to be convinced that real advance had been made. My own impression is, however, that rheumatic heart disease is as prevalent as ever it was. Only a few years ago when I was examining for the University of London at one of our London hospitals, the number of cardiac cases in the hospital was so great as compared with other cases suitable for examination that every one of the candidates, to each of whom two cases were allotted, had to have a cardiac case as one of them.

Nor do I see from a reference to statistics of the medical wards of Guy's Hospital that there is any material difference in the proportion of cases of rheumatism which presented signs of endocarditis or heart disease.

Only a careful review of every individual case would give the information desired in a detailed form; it is sufficient here to say that in the year 1888, 89 cases of acute rheumatism were admitted, and amongst them there were 82 different cardiac lesions in 69 patients (76 per cent.); and that in 1905, 108 were admitted, and there were 116 lesions in 74 patients (70 per cent.)

The cardiac lesions in cases of first attacks were: in 1888, 26 out of 40, or 65 per cent.; in 1905, 23 out of 52, or 44·2 per cent.

The figures are not large enough to compare with advantage. Moreover, in 1888 the salicylic treatment was in force.

In this connection, that is, the effect of treatment, the direct mortality from acute rheumatism is a small matter. The deaths from rheumatism, as is well known, are quite infrequent. In the eighteen years from 1888 to 1905 there were admitted into the wards of Guy's Hospital 1887 cases, or an average of 105 per annum. The deaths from rheumatism during the same years amounted only to twenty, that is, little more than 1 per cent., and they never reached more than two in any given year. (It is true that in one year, 1885, a little before this, there were six deaths.) The number of deaths is too small for the influence, whether positive or negative, of any kind of treatment to be asserted.

There can be no doubt that the great need in acute rheumatism is some method of treatment, whether by drug or serums or whatever it may be, which will not only diminish the fever and remove the pain, but will also save the cardiac tissues from the effects of whatever organism or toxin is concerned.

At different times physicians have expressed the belief that if the cases can be put to bed early enough or sufficiently protected from cold—it has even been suggested that the application of a cool stethoscope to the precordial area is quite enough to determine an endocarditis—that these complications can be kept at bay. Well, these are very difficult things to prove, and for myself I beg leave to entertain the very gravest doubt whether the circumstances which determine the influence of the virus upon the different tissues are so much within our control as these observers would have us

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believe. In any case, even if *theoretically* within our control in primarily articular cases, it is well known that such control only commences when the patient voluntarily claims his assistance, and not always then.

I say nothing of the cases in which the heart is primarily affected ; the damage is already done.

We come then to the further question : Can anything be done for endocarditis as such ?

I leave the question to be answered by others, with the feeling, however, that evidence of the results of each treatment must be submitted to very critical examination.

Another phase of the subject of acute rheumatism, which I may suggest for the consideration of this meeting, is the relation to insurance practice. What view ought to be taken with regard to the liability of a proposer who has had within a few years a definite attack of acute rheumatism, when the heart on examination appears to be perfectly sound? And in these circumstances there are two possibilities, of which the examiner may be cognisant—one that at the time of the rheumatic attack no evidence of cardiac implication was observed ; the other, that endocarditis or pericarditis were on good grounds believed to occur.

The well-known fact that an interval of fifteen or twenty years may intervene between an attack of rheumatism and the onset of cardiac disease cannot be ignored in our consideration of this subject.

Mr. President and gentlemen, I think it is high time that I brought these somewhat rambling remarks to a conclusion. I have, as I threatened, raised several questions, and laid before you few, if any, facts. If it should turn out that to all my questions answers have long ago been given, and that I have simply overlooked them in the concentration of my attention on other matters—and this is possible—then I shall be only too glad to hear them, and I shall regard the disease of acute rheumatism much more favourably than I have hitherto done.

Dr. POYNTON said : I must thank the Council of the Hunterian Society for the honour they have done me in asking me to take part in the discussion, although I confess my position is not an easy one. I am a clinical physician and not a bacteriologist (although fortunately my co-worker, Dr. Paine, is, I think, the greatest authority upon the subject of rheumatism). Then again, I hold with Dr. Paine a view on the subject of rheumatism which the majority of bacteriologists in this country do not hold; and lastly, I am followed by an able investigator who is I believe openly opposed to our conclusions. From the point of view of discussion nothing could be better, but I know too well that it is easier to destroy than build up, and on controversial points it is easier to speak late than early.

To me, rheumatism is a specific disease. Difficult of diagnosis often enough, as are tuberculosis and typhoid, not yet defined but in process of definition, horribly complicated by loose phraseology and crude assumption, but in my opinion a special disease in the accepted clinical sense of that term. As having close bearing on the bacteriology of the disease, I would emphasise that it is not strictly a fever, for there may be no fever. Nor is it necessarily acute, for I have seen many cases in childhood which ended acutely, but showed first a prodromal stage comparable to the early warnings of tuberculosis.

I will not delay you with a detailed history of the bacteriology of the disease. Alfred Mantle was the pioneer in this country, and Triboulet and Coyon in France in 1897 clearly recognised the diplococcus and worked at its chemistry, but believed it to be the cause of complicated rheumatism—an epiphenomenon as it were.

In 1899 Wasserman, Westphal and Malkoff isolated a diplococcus from a fatal rheumatism with chorea and produced polyarthritis in eighty rabbits. The German school, as you are aware, deal largely with a condition called acute rheumatic polyarthritis in contra-distinction to a constitutional disease, rheumatic fever. On account of this, I suppose, these observers were content to produce a polyarthritis, and they relied for their claim upon the investigation of one case.

In 1900 Dr. Paine and I published our first paper, which was followed by various others. With the details I will not trouble you, but will briefly summarise our results up to date.

We have investigated upwards of forty cases in ten years, aided by the kindness of the physicians at St. Mary's, University College, and the Great Ormond Street Hospitals. We have isolated a diplococcus from rheumatic endocarditis, pericarditis, pleurisy, arthritis, a nodule, the brain in

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chorea, the cerebro-spinal fluid, peritonitis, the blood, the urine, and tonsils.

We may justly claim to be the first to have repeatedly produced experimental rheumatic endocarditis and pericarditis, and the gradations from the simple to the malignant forms of endocarditis; also choreiform movements, rheumatic pleurisy, nodules, broncho-pneumonia and peritonitis, and to have first isolated the diplococcus from the tonsil, and reproduced the disease, as also from the nodule. To have produced also acute cardiac dilatation, arthritis, perivascular fibrosis, chronic arthritis, and osteo-arthritis and iritis. You will pardon my putting these points forward, but I am not speaking for myself alone. If the view I put forward to-night proves eventually correct, the elucidation of this constitutional disease as the English conceive it rests with this country.

In the human tissues we have demonstrated it in the valves, pericardium, brain, pia mater, arthritic exudations, nodules on four occasions, blood, pia mater, cerebro-spinal fluid, lungs, pleuræ, and peritoneum.

Before passing on to our conclusions I should like to allude to the works of others in this country on the subject.

Ainley Walker and Beatson made numerous experimental observations upon cases of rheumatism and chorea, but I would especially emphasise the work of Walker upon the acids produced by the diplococcus, notably formic acid. His was the pioneer work on the subject in England, although in France Triboulet and Coyon had made some investigations on these lines.

Vernon Shaw with our cultures was the first to produce the disease in monkeys, and also carried out with me the first investigations on mixed infections with the diplococcus and *Staphylococcus aureus*. He also confirmed the production of iritis and produced relapsing iritis, and the most remarkable chronic arthritis and chronic heart disease I have ever seen in animals. Lastly, he proved that the micrococci isolated by Wassermann, ourselves, and Walker were identical. His work has not received in my opinion due recognition in this country.

Beattie in Edinburgh confirmed many points, including the production of choreiform twitchings. He proved also the existence of the infective agent in the human synovial membrane when the arthritic exudation was sterile, and made valuable investigations upon chronic renal changes, experimentally produced in rabbits.

No one has worked more steadily for the proof that the micrococcus is specific, and if from a natural caution I still feel the proofs are not conclusive, it is not from a lack of appreciation of his investigations in this direction.

Gordon Holmes, working with me in the investigation of rheumatic chorea, has entirely, from his own part in the research, placed that condition upon a reasonable and working anatomical basis.

In America Longcope's confirmation of the fact that the exudation in experimental rheumatic arthritis may be sterile has scientific bearing upon the disease.

Fritz Meyer's extensive work on rheumatic angina faucium stands out prominently in the German literature, as does the work of Allaria in the Italian.

Next as to our conclusions, and the reasons for them. With these I will be brief. They are included in two statements and in one expression of opinion.

The statements are—

(1) A diplococcus is a bacterial cause of acute rheumatism.

(2) Rheumatism may be a cause of simple and malignant endocarditis.

The expression of opinion is that because no other micro-organism appears to satisfy the necessary postulates the diplococcus is the only bacterial cause of the disease.

This opinion leads me to speak of the large anthrax-like bacillus described by Achalme and claimed by Thiroloix as a cause of rheumatism. We have never found it, and for some while all evidence in its favour had failed. Now again recently Thiroloix and Rosenthal, in 1907, have isolated it from the blood in virulent rheumatism and make a new statement to the effect that this anthrax-like bacillus outside the tissues in culture will become a diplococcus. Possibly it may, but all we can say is that we have not found the bacillus, although the diplococcus in unsuitable media out of the body may become bacillary, and in fact take almost any shape or form.

I want to lay particular stress upon the reasons for our conclusions, because these have been somewhat distorted. They are:

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(1) The micrococcus can be isolated in pure culture from the cardinal lesions of rheumatism.

(2) It can be cultivated in pure culture.

(3) It can produce by intra-venous inoculation similar lesions in animals.

(4) It can be isolated from these lesions in pure culture.

The specimens kindly lent me by the College of Surgeons, which are some of our original ones, illustrate some of these points, and drawings I have with me from the preparations we have shown before societies illustrate others for those who desire visible proof. I hand round cultures of the diplococcus, not because they are of convincing importance but as proof of its existence.

I don't wish to fatigue you with irritating details about the diplococcus. It belongs to the streptococcal group. It is minute and resembles a cut-down pneumococcus without a capsule. It grows best in diplococcal form, and is very resistant to drying. The virulence, easily lost, is best preserved on blood agar. It is strongly acid-producing.

Beattie lays stress upon the power of producing acid and precipitating the bile salts in McConkey's bile-salt lactose broth.

I shall not withdraw a single point from these conclusions, but reply now to some of the chief objections that have been raised.

The first is the failure on the part of skilled bacteriologists to find this micrococcus. I can only point to the positive results obtained by ourselves and others, and claim that we have shown our methods.

The second is the statement that the infection is a terminal infection. I can vouch for a patient still living and working some years after the micrococcus was isolated from the blood during an acute attack of pericarditis. Another case of arthritis certainly left the hospital, as also a case of chorea. Perhaps, however, the most convincing case was one under Dr. Lees, which lived a month after isolation of the diplococcus. The post-mortem showed the case was one of severe rheumatism.

Experimental investigation also disposes of the objection, which I hope will now be dropped as untenable.

The third objection is important, but our statements upon the point have been frequently misrepresented.

It is to the effect that the experimental results are in no way peculiar, but are caused by various micrococci. We lay much stress upon the results. No micro-organism that we have worked with has produced heart disease so often—an independent experience of Beattie's also. But our point is this—the results were produced by a micrococcus obtained from cardinal lesions of rheumatism in pure culture. *That is the crucial test.* We all know there are many causes of heart disease and arthritis in man which are not rheumatic, those produced by the pneumococcus, the influenza bacillus, the gonococcus, etc. The same is probably true in animals, and we can vouch for it with the *Staphylococcus aureus*, and with streptococci other than the rheumatic. With the staphylococcus there occurs also pyæmic abscesses which we have never found in rheumatic lesions, and in our hands the streptococcus so-called *pyogenes* only produces endocarditis rarely. And even granting this were frequent, it does not prove that the toxins are identical, and until we know more of the toxins I see no way of proving the point one way or another. It is unlikely, however, that every organism that produces an endocarditis does it by an identical process.

It is surely an evident necessity in any investigations upon rheumatic endocarditis, for example, that the micrococcus should be isolated from a rheumatic lesion.

Lastly, it has been said that it was impossible to recognise the diplococcus from our description. Triboulet, Wassermann, Ainley Walker, Shaw, and Beattie recognised it from our description as did we from theirs, and Shaw showed their identity. I may add that Gustav Singer, the Austrian investigator, recognised it when we were at the International Congress in Madrid. I am unable, then, to accept this objection, but admit that we gave no striking specific laboratory test, because it was quite clear that we were face to face with one of the most difficult problems in bacteriology—the streptococcal group of micro-organisms.

There are very definite difficulties connected with the bacteriology of rheumatism. The chief are:

(1) The minute size of the diplococcus.

(2) The fact that rheumatism consists of numerous local lesions, and although the blood is a channel of conduction this is only in exceptional cases a probable source from which to isolate the micro-organism.

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(3) The rarity with which fatal rheumatism occurs. Acute pericarditis affords the great opportunity, and it is very exceptionally that one fails to demonstrate the diplococcus in films, in sections, and on culture. It is, however, for example, months since I have seen a fatal case.

(4) Unfortunately the virulence of the micrococcus rapidly diminishes. It grows most sturdily. This culture I am showing is a descendant from an original of ten years ago, but it is not virulent now however enormous the number injected.

(5) When we consider the local lesions, it becomes clear that the micrococcus does not easily escape into the exudations, and if it does is probably soon destroyed.

This was a great puzzle at first, and particularly with regard to arthritis, but now it is worked out experiment shows that in acute arthritis the exudation may be sterile, and Beattie proved our hypothesis that the micrococcus was in the synovial tissues of the human joint when the exudation was sterile.

There are the same difficulties to a lesser degree with pericarditis and with healing simple endocarditis. When I add that we worked a year before we could find the micrococcus in the tissues it is clear that the difficulties are very real.

(6) The greatest difficulty of all, and one which appeals to practical minds, is that of getting some reliable laboratory test. This problem—the laboratory differentiation of streptococci—many are working at, each in his own way. To me it seems essential at the present time to use methods that maintain the virulence of the particular streptococcus. Once lose that and all other chemical changes it may produce appear to me of secondary importance.

For example, various members of the same group of micrococci may ferment the same sugars, but it does not follow they produce the same diseases in the body. These diseases are probably the results of highly complex toxins of albuminous nature.

So much has been demanded by bacteriologists from what they term a "specific" micrococcus, that when we bear in mind that in this country quite a short time ago the word "streptococcus" meant the *Streptococcus pyogenes*, whereas now, with their strains and groups, bacteriologists are admitting that their dicta are not so certain as they thought, we cannot wonder that the laboratory test is slow in appearing.

Let me ask you, lastly, for a few moments to accept rheumatism as a diplococcal infection, in which the particular soil is probably of much importance, and follow some of the lines of thought that are opened up.

The great infections we are acquainted with vary in virulence, in type, and in the personal reactions they call out. Analogy, I admit, may be dangerous, but it is an essential in investigation. Consider miliary tuberculosis, broncho-pneumonia, tuberculosis, phthisis, and chronic fibroid phthisis, and then take one rheumatic lesion only—the articular. Why should not this infection produce synovitis, subacute periarticular arthritis, and osteo-arthritis? Why should a dogma that rheumatic arthritis and acute gout have nothing to do with rheumatism cramp inquiry? Can anyone here give a better explanation of some acute gouty attacks than this—that in a gouty soil the acid-producing diplococcus of rheumatism has got access and produced locally in the gouty tissues biurate of soda? I don't say it is correct, but it is quite as probable as any other view, and, in my opinion, rather more. Why should we accept on no convincing evidence that rheumatism and salicylate of soda are hook and eye?

We have no accurate knowledge of the toxins, and no right to assume that they, the same, are produced in the different tissues.

Lastly, as to bacteriological lines of treatment. For some years I have been trying sera, but with no clear success; and in the present state of medical opinion it would be little short of a crime to publish anything but the most convincing results.

The opsonic investigations are being carried out, and we all must hope for the success of a vaccine, but I hesitate at present to rush headlong like the Gadarene swine over the precipice.

Dr. HORDER spoke on the bacteriology of allied conditions. He said: I thank you, sir, for your kind invitation to take part in this discussion. Dr. Poynton has referred to me as being one of those who are openly opposed to his views, or to the views of himself and Dr. Paine, on the ætiology of rheumatic fever. But I should prefer Dr. Poynton

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to believe that my attitude is one of a sceptic rather than of an opponent, because I really am honestly following—it is true at a distance—in the footsteps of Dr. Paine and Dr. Poynton and of others who have worked on similar lines to these investigators. But there is this distance at present which I have not yet been able to make up. And my own remarks this evening will refer to the difficulties I have, which make me halt a little in the race. I can perhaps best refer to certain results of my own connected with the bacteriology of rheumatic fever and of that very closely associated condition or disease, a careful study of which throws a flood of light upon the ætiological problem of rheumatic fever. I refer to "ulcerative" or "malignant" endocarditis. I shall refer only to my observations in connection with the bacteriology of the blood-stream in these diseases; I have not as yet a sufficient body of evidence or statistics with regard to the investigation of joint fluid or cerebro-spinal fluid or serous effusions to make statements with regard to these. I have investigated the condition of the blood during life in thirty cases of undoubted *rheumatic fever*, choosing well-marked cases, in which the diagnosis has admitted of no doubt. Endocarditis has been present in most of the cases, and in some of them there has also been pericarditis and chorea. From these cases I have not been able to isolate any micro-organism which I could think with reason came from the blood-stream. I say this because in the course of blood-culture work one is occasionally faced with a positive growth which, when investigated further, proves to be a contamination. I refer particularly to skin staphylococci. I have conducted post-mortem investigations in some sixteen cases, along similar lines, not only dealing with the blood, but with pericardial effusions, pleural effusions, and with vegetations from the valves. From five of these cases I have isolated micro-organisms which were not contaminations. And they have in all cases been streptococci, not in any large numbers, and possessing no special features. In each of the five cases the strains of streptococci have been such as are met with frequently in post-mortem investigations conducted on patients dead of non-rheumatic diseases. In other words, my results have been, from the point of view of the isolation of a specific micro-organism, entirely negative. And I have used for these blood-culture experiments quite a variety of nutrient media, including the particular medium which Drs. Poynton and Paine suggested was one for which the "*Diplococcus rheumaticus*" has a particular affinity; I refer to a medium consisting of equal parts of milk and broth rendered slightly acid by the addition of lactic acid. I have invariably used as much as 5–10 cc. of blood for the cultures.

Turning to "*malignant endocarditis*," my results in that condition have been just as strikingly positive as they have been negative in rheumatic fever. Out of 33 cases which were proved to be ulcerative endocarditis, either clinically or post-mortem, in 28 I have obtained positive results; not always at the first attempt in the culture investigation, but sometimes at the second or third attempt. My post-mortem investigations of malignant endocarditis cover more than sixty cases, but of those I do not choose to speak, because of course any micro-organism obtained from the blood-stream during life must be, so far as this question of the causal factor is concerned, of much greater importance. Of these 28 positive cases, in 18 the micro-organisms have been streptococci. And of the 18 strains of streptococci found, 16 have been short streptococci. A large number might even be called diplococci, "*Streptococcus brevis*" as it is sometimes called, or following the nomenclature for which Dr. Andrews and myself are responsible, the large majority of these streptococci have been of the types of *Streptococcus salivarius*, and *Streptococcus fæcalis*. We merely indicate by these names that there are types of short streptococci, the source of which is the alimentary canal, either the mouth or the intestine, which may become pathogenic, although the degree of pathogenicity may be very feeble, as witness the very chronic nature of some of these cases of malignant endocarditis. Becoming pathogenic the streptococci are prone to pitch upon diseased heart-valves—valves which have become diseased in the majority of cases by previous rheumatic fever. But not in all instances by rheumatic fever; in some of the cases there has been no history of rheumatism, but there has been a definite history of syphilis, or of gonorrhœa, with resulting endocarditis. In some of the cases there is no acquired valvular disease, but the cardiac condition is congenital. Now it so happens that such of the specimens of the *Diplococcus rheumaticus*

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as Dr. Andrews and I have been able to investigate on biological lines have all of them given reactions identical with the reactions of the streptococci obtained with such constancy from cases of malignant endocarditis, and especially of *Streptococcus fæcalis*. Experimenting with these short streptococci obtained from patients suffering from "malignant endocarditis," I have been able to obtain results in rabbits which have struck me as being, if not identical with, then closely analogous to, those obtained by Drs. Poynton and Paine when using their *Diplococcus rheumaticus*. I have produced, by means of these short streptococci, endocarditis, pericarditis, and in one case a certain amount of multiple arthritis without suppuration. Following up this experimental work, I thought it would be interesting to see whether the so-called "saprophytic" streptococci from the alimentary canal could be made to produce in the rabbit the lesions of endocarditis and pericarditis. And I found that that was easily done. I found that short streptococci, isolated from normal stools and from normal saliva and injected into the blood-stream of rabbits, does produce in two or three weeks a very definite and fatal form of endocarditis. True, the endocarditis set up was always a fungating endocarditis. But I believe, though I am open to correction, that the endocarditis set up in rabbits experimentally by the *Diplococcus rheumaticus* has usually been of the fungating variety. Certainly the photographs of such rabbits' hearts appear to prove this.

Such are my facts, very briefly put. Time does not allow me to deal at length with the interpretation of them. But these facts seem to me to admit of a possible explanation of the discrepancy which it is only fair to say exists between the positive results of bacteriological investigations in rheumatic fever of Drs. Poynton and Paine and Dr. Ainley Walker on the one hand, and of the negative results of Drs. Bullock and Andrews and myself on the other hand. The possible explanation is this: that the cases from which the *Diplococcus rheumaticus* has been obtained during life and post-mortem have been cases of ulcerative endocarditis. It is at least suggestive that Drs. Poynton and Paine recently coined the name "malignant rheumatism" to cover a set of cases from which they isolated the *Diplococcus rheumaticus*. It certainly is a striking fact that the chief micro-organisms present in ulcerative endocarditis, *whether the patient has suffered from rheumatic fever or not*, are micro-organisms which, so far as the most searching differential tests of bacteriology can tell, are identical with the short streptococcus to which the name *Diplococcus rheumaticus* has been given. It is also a striking fact that by tracing this short streptococcus back to its source in the human intestine it can, by isolation from this source in the healthy person, be made to set up results in rabbits which are strictly comparable with the results obtained by Drs. Poynton and Paine when working with the *Diplococcus rheumaticus*. In concluding, I cannot help drawing your attention to the close analogy there appears to be between the problems of the ætiology of rheumatic fever and that of scarlet fever. In scarlet fever pure and simple, we still await convincing evidence with regard to the bacterium concerned, if, indeed, it be a bacterium. But in "malignant" scarlet fever, that is to say, in scarlet fever *plus* certain highly dangerous and often fatal complications, the ease with which micro-organisms are isolated from the patient (and these micro-organisms are nearly always streptococci) is a very noteworthy fact. I believe the causal factor of rheumatic fever is as yet undiscovered, and that such bacteria as have been obtained from "rheumatic" patients are connected with secondary invasions of the body by streptococci which obtain a nidus in parts damaged by the rheumatic virus. Whether the specific syndrome we term "rheumatic fever" is an acute intoxication of the blood by absorption of poisons from streptococci existing in the alimentary tract,—streptococci, which only in the graver cases, and especially in those developing fungating endocarditis, succeed in overstepping the barrier of the tissues and invading the blood-stream—remains a question for further investigation.

(*To be continued*).

**Secretions of the Suprarenals in the Blood and Urine.**—Waterman and Boddaert say that the presence of adrenalin in the blood and urine of nephritics is not at all proven. Before making any conclusions as to the *rôle* of adrenal products in nephritis a good test for the substance must be found.—*Med. Record*, vol. lxxiv, No. 3.

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# ON PUERPERAL FEVER.*


By VICTOR BONNEY, M.S., M.D., B.Sc.Lond., F.R.C.S., M.R.C.P.,

Assistant Obstetric Physician to the Middlesex Hospital, Surgeon to Out-patients, Chelsea Hospital for Women; Hunterian Professor in Pathology, Royal College of Surgeons of England.


GENTLEMEN,—I propose to put before you this evening a few facts concerning the ætiology, pathology and symptoms of puerperal fever, and in their light to pass on to a consideration of its cure and prophylaxis.

The subject, though well worn, is both interesting and important, for this disease, although practically extinct as far as lying-in hospitals are concerned, is still a far too common disaster in the case of women confined at their own homes. Indeed, the number of cases reported from this source shows singularly little diminution, and this in spite of the fact that antiseptic precautions of some kind or other have been exercised in the large bulk of home-conducted labours for many years.

*Ætiology.*—I will begin, then, by reminding you that the cause of the disease is well known, and the idea that it was due to some specific organism of mysterious powers and all-pervading properties has long since been exploded. Pasteur isolated streptococci from these cases many years ago, thus indicating the relation borne by the disease to ordinary wound sepsis and simplifying our conception of its nature. Unfortunately the work of certain German bacteriologists tended somewhat to re-mystify the medical public, for whilst ascribing a proportion of the cases to infection by the ordinary flora of wound sepsis they separated off a so-called "sapræmic" group whose existence was said to depend upon certain anaërobic organisms. The exact nature and properties of these anaërobic organisms they did not define, and there is reason

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to believe that they were, in many cases, merely cultural contaminations bearing no relation to the disease in question.

As a result of an investigation of the bacteriology of puerperal sepsis carried out by Mr. A. G. Foulerton and myself, it was found that in the great majority the disease was attributable to the organisms of ordinary septic infection. We separated our cases into three groups: (1) Those with severe symptoms terminating in death; (2) those with severe symptoms ending in recovery; and (3) those with slight symptoms.

We found that each of these groups exhibited peculiar features as regarded their bacteriology.

Thus in the first group a dual infection was almost always present, viz. some variety of streptococcus combined with the *B. coli communis*. On one occasion the pneumococcus took the place of the streptococcus, and in two cases the streptococcus was alone present.

In the second group the streptococcus was again predominant, though the pneumococcus was isolated on several occasions. The striking difference between this group and the last was that the infecting organism was nearly always found in pure culture and not combined with the colon bacillus.

In the third group the uterine cavity was sterile in a considerable number of cases, but in others it contained staphylococci and *B. coli communis*, either singly or in association. But in all the cases of this group these organisms were plentifully found in the vagina and cervix. It therefore appeared that: (1) The presence of the colon bacillus in the uterus in conjunction with streptococci or pneumococci was of evil import, and (2) that in a fair number of the slighter cases the symptoms are due to vaginal or cervical absorption.

The colon bacillus is probably a secondary infection in most cases of puerperal sepsis, and its presence appears to accentuate the symptoms to a marked degree.

In the vast proportion of cases, then, the disease is due to the ordinary organisms of wound sepsis. How do these find entrance into the normally sterile uterus? It has been shown that ordinarily the vagina does not contain the more virulent organisms. Even the cervix in chronic cervicitis was found by Mr. Foulerton and myself to be free of them. On the other hand the staphylococci (and the gonococcus, of course) are often present in the cervix and vagina. The colon bacillus, like the poor, is always with us, if not actually in the genital passage at least in very close proximity to it, and it is a well-known fact that damaged tissue in close proximity to the bowel very soon becomes infected by this organism.

Thus puerperal sepsis is both heterogenetic and autogenetic; heterogenetic in most cases of streptococcic and pneumococcic infection, but very often autogenetic as regards the infection by the staphylococci, or the colon bacillus.

There is, however, a second factor in the production of any infective disease which is no less important than the infecting organism itself, viz. the resisting powers of the tissues, and it is of importance to remember in this connection that besides a constitutional susceptibility to pyogenic organisms as shown by a low opsonic index, there is also such a thing as local susceptibility, in the causation of which trauma is the most important factor.

I will now pass on to a consideration of the symptoms of puerperal fever, and at the outset I would remind you of the extreme variability of the clinical picture presented to us by these cases. This is in part due to the varying causal organisms, but it is also due to the different lesions present in different cases.

The lesions of puerperal fever may be divided into three: (1) The primary lesion, (2) the consecutive lesions, (3) the generalised lesions.

*The primary lesion* is situated at the placental site in all the more severe cases, but in some of the slighter forms lacerations of the cervix or vagina may be first or solely affected. The condition of the placental site differs in different cases. In pure streptococcal cases it is usually covered by a greyish-looking diphtheroid slough, and it is in these cases particularly that the lochia are apt to be suppressed, for you cannot get blood from an area of hyaline necrosis. In cases associated with the colon bacillus and streptococcus combined, the surface is brownish or greenish, and of a pultaceous consistence. The discharge in these cases is brownish or yellow, and very offensive.

In a few streptococcal and all severe staphylococcal cases (*Staphylococcus aureus*) the area of placental attachment is in a condition of frank suppuration, and the discharge is pure pus.

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The uterine wall itself is swollen and œdematous and involution is inhibited, indeed, the uterus may actually increase in size owing to this swelling. When explored by the finger the placental site has a peculiar lobulated surface, and feels soft and "greasy." In a few cases actual collections of pus may be found in the musculature and under the peritoneal coat.

*The consecutive lesions* are many, for starting from the placental site, the infection may spread to the Fallopian tubes, the peritoneum, the cellular tissue of the broad ligaments or the large veins contained therein.

As regards peritoneal infection it is important to remember that in some of the worst cases there are none of the classical signs of peritonitis present. *Post-mortem* we find scarcely any redness of this membrane and no adhesions, but the pelvis contains a blood-stained serum, which under the microscope is found to contain as many streptococci as a twenty-four-hour bouillon tube laboratory culture. These are examples of infection without inflammation, for so over-weening has been the bacterial growth that the tissues have been overpowered before they could react protectively. Such cases are always fatal.

In other cases, however, varying degrees of peritonitis may be present. Those in which it is localised to the pelvis and in which suppuration occurs are the most likely to recover, for pus formation is an evidence of tissue resistance. Localised peritonitis usually produces a definite pelvic swelling after a while, whereas there is no tumour formation when it is generalised. In these cases of localised suppuration pus is always found exuding from the tubes, and it is by this route that the peritoneum became infected.

The cellular tissue of the broad ligaments when affected is œdematous and swollen. In particular there is a phlebitis of the broad ligament veins, which are thrombosed and may even contain pus. In some cases this thrombosis extends to the external iliac and femoral veins, and the leg swells. This happens almost invariably on the left side for reasons that are unknown to us.

In some of the more chronic cases the parametric inflammation produces a definite tumour, but in the graver cases no swelling is to be distinguished, for as in the peritoneum it is a case of infection without inflammation.

*The generalised lesions* are those due to the presence in the blood stream of the toxic products of the infecting organism or of the organism itself. They are therefore always *toxæmic* and may be *septicæmic*. The presence of these bacterial toxins in the blood plasma causes diffuse degeneration of the cells of the organs in which it circulates. These lesions are generally known as "the cloudy swellings," and if long continued they terminate in a definite fatty degeneration.

Thus the liver, spleen and kidneys are found to be swollen and soft and in their latter stages are pale in colour, and heart muscle and nerve cells suffer correspondingly.

When there is an actual blood transportation of the organisms (septicæmia) we have in addition to these toxæmic lesions localised areas of secondary infection and inflammation, and since organisms most commonly find their way into the blood stream by the medium of thrombosed veins, it is obvious that the lungs are the most likely organs to become secondarily infected. Thus in cases that die evidence of septic pneumonia is frequently present. But the organisms may pass through the pulmonary capillaries, and being deposited in the liver, spleen, brain or elsewhere set up local areas of inflammation in these situations. When the patient lives long enough, and when her tissues are sufficiently vigorous, these areas of secondary inflammation suppurate and the term "pyæmia" is applied to the condition.

For a patient to die it is not necessary, however, that generalised *septicæmic* lesions should be present. They may be purely *toxæmic*, the organisms remaining local at the site of the primary lesion, or of the primary lesion and consecutive lesions combined.

A consideration of these points in the pathology of the disease will, I hope, make clear to you the reasons for its varying types. Just as a septic infection of the finger pulp may lead in one case to a local whitlow, in a second to a whitlow plus a diffuse cellulitis of the arm, in a third to a diffuse cellulitis with scarcely any manifestations in the finger, in a fourth to suppuration of the axillary glands, and in a fifth to general septicæmia, so infection of the placental site in one instance may give rise to uterine symptoms only, in another to these plus signs of peritonitis and cellulitis, singly or combined, in another to severe septicæmic

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symptoms with few uterine signs, and in another to a combination of all of these together.

It is a want of appreciation of these facts that leads to such diagnostic errors as mistaking the symptoms of puerperal fever for those of influenza, scarlet fever, pneumonia, and so on. Moreover, one must admit that there is a natural dislike in all of us to believe that any case under our care has "gone septic," and a wish to find some other explanation for the symptoms, and the wish, as we know, is often the father of the thought. In the case of puerperal pyrexia we should reverse the famous principle of English law which assumes innocence until guilt is proved, and hold every rise of temperature in the puerperium as being due to sepsis until there is positive evidence that it is not so.

### The Treatment of Puerperal Fever.

It is, however, on the treatment and prevention of puerperal fever that I want to lay most stress this evening, and it is to this end that I have endeavoured to present to you the picture of the disease as it appears to the mind of the pathologist.

And I think that if you see eye to eye with me in this matter you will agree that the first lesson which pathology teaches us as regards the cure of the disease is the importance of rapid action on our part, and the gravity of losing time in the application of our curative measures. For the successful attack of this disease it may be truly said that each hour counts. For it is almost certain that every case, were it recognised and treated early enough, would be saved. Unfortunately this is not so. It is my lot to have seen a number of these patients, and in the bulk of them no active interference has been meditated until several valuable days have elapsed.

All fever in the puerperium should be regarded as septic unless it can be proved otherwise. *A temperature of* 100° F., *if it has existed for twenty-four hours, should be considered as an indication to act. A temperature of* 101° F. *or over should be regarded as an immediate indication.*

It is true that by adopting this rule a good many cases are operated upon which would no doubt get well without interference, but this is a matter of small importance in comparison with the saving of life effected by the early treatment of the remainder.

*Uterine exploration.*—The first thing to do in any early case of puerperal sepsis is to explore the uterus under anæsthesia. I have reminded you that in some of the slighter forms the uterine cavity is sterile, and this fact might be held to contra-indicate exploration of the uterus in some cases. But we have no sure means of rapidly ascertaining whether the uterus is or is not infected, and it is better therefore to treat all cases on the assumption that they belong to the graver category.

No harm in my experience ever follows uterine exploration when properly carried out, but irreparable ill may result from delay.

The patient should lie on her back in the long axis of the bed. It is a mistake to place her on her side or in the lithotomy position, because in either of these it is much more tiring to make strong pressure on her abdomen.

This strong pressure is a necessity to force the uterus on to the exploring fingers, for without it the longest fingers are unable to reach the top of the cavity.

It is an advantage to place her on a bed pan, or better still, a douche bath, so as to avoid wetting the bed, and I find it convenient to sit on the right edge of the bed facing the patient whilst carrying out the operation.

The whole of the gloved right hand without the thumb (unless there is room for it) is inserted into the vagina, and one finger, and subsequently two, are passed up the cervix into the cavity of the uterus. To efficiently explore the uterus, and certainly to remove anything from its cavity, two fingers are a necessity. It often happens that the cervix at first feels too small to admit of more than one finger, but after a few minutes it will be found that a second can almost invariably be introduced. The uterus is firmly pressed down by the left hand on the abdomen, and it is now that that the advantages of the position I have described are felt, since one is able to make use of one's weight instead of relying only on muscle effort. The whole of the surface is explored, particularly the placental site, which is roughly scraped by the gloved finger-nails. Small shreds and fragments of placental tissue, decidua or blood-clot can always be extracted from any recently delivered uterus. At times our search is rewarded by the finding of larger masses. By a movement of the

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fingers in the cavity the fragments are passed through the cervix into the palm of the hand in the vagina. When the evacuation is complete the fingers are withdrawn through the cervix and the intra-uterine douche tube passed into the uterus, which is then washed out with two or three quarts of a 1 in 2000 solution of biniodide of mercury at a temperature of 120° F. The tube being withdrawn and all fluid having been squeezed out of the uterus, the external genitals are cleaned up, soiled linen and douche water is removed, and the patient is allowed to come round from the anæsthetic.

The effect of this operation, when performed early, is most satisfactory, the temperature almost immediately falling. In a few cases, however, an abrupt rise of temperature accompanied by a rigor ensues. This, though alarming, is followed as a rule by a rapid fall to the normal, but it is worth while mentioning its possibility to those in attendance on the patient. It is difficult to determine why uterine exploration acts so satisfactorily. Probably the reasons are as follows: (1) The evacuation of infected fragments and clot; (2) the improved drainage of the cavity that the dilatation of the cervix effects; (3) the increased tonicity of the uterine wall brought about by the manipulations and the hot douche.

### Curettage of the Uterus.

Exploration of the uterus in the manner I have detailed is almost invariably successful if carried out at the onset of the disease. In later cases, however, its results are less beneficent, and the longer the period that has elapsed since the onset of the disease the more inadequate does this treatment become. These late cases may be divided into three classes: (1) Those in which curettage of the uterus is indicated; (2) those in which abdominal or vaginal section is advisable; and (3) those in which no operation should be undertaken.

Curettage of the uterus is an old treatment which had fallen somewhat into disrepute but which has recently been strongly advocated by Knyvett Gordon, who has published a very important paper on the subject. In this he compared the results obtained from a series of cases treated by this method at the Monsall Fever Hospital with a similar series treated by the more accepted methods, viz. exploration and serum. His figures showed a marked superiority for the treatment by curettage. It is to be remembered that the cases of puerperal sepsis admitted into a fever hospital are almost all of a serious and advanced type; yet, in spite of this, he was able to record a large percentage of recoveries.

His method consists in curetting thoroughly the whole cavity of the uterus with a very large sharp curette, and subsequently rubbing into its wall pure "medical Izal."

Izal, according to Knyvett Gordon, has a selective action on necrotic and sloughing tissue, and it was for this reason that he originally used it. The Izal having been applied the cavity of the uterus is packed with "Izal gauze," or with gauze wrung out in a solution of Izal.

To perform this operation efficiently the patient must be placed in the lithotomy position by a Clover's crutch, and a duck-bill speculum and an assistant are required. The anæsthetic is a matter of much importance, because the operation takes some time, and where the condition of the patient is grave Knyvett Gordon speaks favourably of performing it under analgesia produced by morphine and alcohol in the manner lately recommended by Sir W. Japp Sinclair.

I have performed this operation myself but not often enough to form a judgment of its value, but the evidence adduced by its author appears to me to warrant its being given the fullest trial. I have found that when using pure Izal care must be taken not to burn the patient's vagina, vulva, or thighs, and it is scarcely necessary to remark that the proper performance of the operation, especially when dealing with a totally subinvoluted uterus, requires much more operative skill than the simple exploration.

### Abdominal and Vaginal Cœliotomy.

In cases in which the infection has spread to the peritoneal cavity, treatment directed simply to the uterus is obviously insufficient. These cases have a very bad prognosis in particular, because it is so difficult to diagnose peritoneal infection, since in many of the worst cases, as I have pointed out, the classical signs of peritonitis are wanting. Pain is never a prominent feature of these poor patients, nor do they vomit much as a rule. Rather should one look for signs of slight or moderate distension of the intestines combined with immobility of the

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lower abdominal parietes and tenderness in this region together with a great augmentation of the constitutional symptoms.

Up to the last few years such patients have been regarded as practically hopeless, but lately Knyvett Gordon, to whose work I have already referred, has published a number of cases in whom recovery appears to have been directly due to operative interference. In all cases in which peritoneal involvement was suspected and the patient's condition warranted it he opened the abdomen. He found, as a result, that in these cases there is a direct out-pouring of pus from the tubes into the peritoneal cavity. These were ligatured off and an opening into the vagina was made through which the pelvis was drained, as well as by a large tube passed through the abdominal wound.

The general peritoneal cavity was not irrigated, the pelvis being simply mopped out and the whole operation was conducted as rapidly as possible. The results attained by Knyvett Gordon are so encouraging that his procedure will undoubtedly be generally tried.

Drainage of the pelvis by a vaginal incision has been practised by many surgeons with indifferent success, for it is obvious, considering the state of the tubes, that this operation is inadequate to remedy the lesion.

### Hysterectomy and other Operations.

Removal of the septic uterus has been carried out both by the abdominal and vaginal route, with the result of clearly proving that this course is never to be recommended, for almost all the patients have died.

I will also remind you that parametric collections of pus may be successfully drained by abdominal or vaginal incision, but that few cases present so circumscribed a lesion to operate upon. Lastly we may remember that cases of broad ligament phlebitis have been successfully dealt with by ligature of the veins there, particularly that large leash which accompanies the ovarian artery on each side.

### Cases in which no Operation is Advisable.

There are certain cases in which it is inadvisable to undertake any operation. Where the general condition is grave, the temperature high and fluctuating and associated with multiple rigors, and yet withal the uterus is neither abnormally large or painful, the abdomen not distended nor tender, and the discharge such as it should be, it is probable that a generalised septicæmia is already present, and an operation under such circumstances will do no good, and may even extinguish the last chance the patient has remaining to her.

Much judgment is often required to rightly determine whether an operation is or is not to be performed. Each case must be dealt with on its merits and no rule of universal application can be laid down, but, in general, it may be said that the longer the time that has elapsed since the onset of the symptoms the less likely is an operation to do any good.

### Non-operative Measures.

I will now pass to a consideration of those curative measures that do not fall under the head of operations.

*Intra-uterine douching.* — This is so time-honoured a treatment of the septic puerperal uterus that I feel very diffident in decrying it. Nevertheless, in my experience I have found it a singularly disappointing proceeding. To begin with it is not an operation that it is always easy to carry out. With a restive patient it requires a great deal of manipulation, and this frequently gives considerable pain. When the tube is introduced fairly up to the top of the uterus it is still very questionable if the fluid reaches all the interstices of the cavity, for I believe that the heavy collapsed walls of the non-involuted organ frequently compel the fluid to run straight back in the track of the tube without effecting a thorough irrigation. At the most it can only wash out the organisms loose in the cavity or affect those superficially placed in the wall, whereas the patient's symptoms are chiefly due to those more deeply placed. But perhaps the main reason why I have so poor an opinion of this method of treatment is the frequency with which I have seen time wasted on it to the fatal postponement of the radical operative measures I have described to you. If it is carried out at all it should be *after the uterus has been explored*, but I must confess that I have never seen improvement which could without doubt be attributed to it under these circumstances.

*Serum therapy.*—Though the results of serum

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therapy in this disease have not been as good as might have been expected considering its known bacterial cause, yet it should always be undertaken. Seeing that the streptococcus is much the commonest organism present in the uterus, anti-streptococcic serum should be administered directly after exploration of the uterus has been carried out. During this operation a portion of the uterine contents should be immediately placed in a test tube (sterilised by boiling), and sent to a reliable bacteriologist with a request for a report as soon as possible. For those who possess some knowledge of the microscopical aspect of organisms, and have the necessary oil-immersion lens, an immediate examination of a coverslip smear stained by methylene blue may be undertaken. The nature of the infecting organism having been definitely ascertained, the correct serum should be immediately obtained and administered. Anti-streptococcus serum should be given in much larger doses than is commonly used, viz. 20 c.c. at the first administration, 20 c.c. six hours later, and 10 c.c. six hours after that, making 50 c.c. in twelve hours. If no good effect is produced by this dosage it is certain that that serum is not the correct antidote for the infection in question, and another should be tried.

I need not say that a polyvalent serum is preferable to one derived from the action of a single species of streptococcus, for as you know there are many varieties of that organism, but I would point out that inasmuch as Mr. Foulerton and I found that the co-existence of the colon bacillus with the streptococcus was of very bad import in cases of puerperal fever, it would appear to be rational to give a dose of anti-colon serum as well as the anti-streptococcic serum, and that not only in cases where the uterus contains the former organism, but as a prophylactic measure in all others. I always make a practice of doing this.

*Vaccine treatment.*—There is another method of treating these cases which is so entirely modern that it has scarcely been put on its trial, viz. the administration of a vaccine. In order to prepare this it is necessary to isolate the infecting organism, and subsequently to make a sterile bacterial emulsion, which is injected into the patient in appropriate doses. The work of Sir A. Wright has shown us that the immediate effect of such an injection is to reduce the opsonising power of the blood-serum (negative phase), but that within a short time a reaction occurs, and the opsonic index rises to much above the normal, and with it the resisting power of the patient (positive phase). The beneficial effect of this treatment in furunculosis and some other diseases due to pyogenic bacteria is admittedly remarkable, and it certainly deserves a trial in puerperal fever, particularly in those cases in which the disease runs a long suppurative course. The details of the treatment must be left to a competent bacteriologist, but the practitioner must render it possible by forwarding to him as soon as possible the contents of the uterus as obtained rather by exploration or by passing a sterile swab into the cavity.

### The Prevention of Puerperal Fever.

Finally I would venture to make a few remarks on the prevention of this disease, the continued existence of which has been held up, most unjustly I think, as a reproach solely to the medical profession. That our profession is hardly dealt with in this matter is best demonstrated, I think, by the fact that in the lying-in hospitals, where the will of the doctor is supreme, it no longer occurs.

*The responsibility of the public.*—I myself hold that the public is mainly responsible for it, and on two accounts, uncleanliness and niggardliness. In regard to the first I need not expatiate before an audience of medical men. Which of us has not confined women in a room so filthy that the very odour of it on entering makes the stomach heave, where all the available water is contained within the compass of a small kettle, where the bed and body linen of the patient is brown with the exudations and exhalations of ages, and of which the bug and flea are normal denizens? It is much, I think, to the credit of the doctor that the vast bulk of confinements, including many difficult cases, are carried out under such insanitary circumstances with satisfactory results to the patient. Personally I never go into such a room as I have described without marvelling that in this twentieth century the law permits such places to be used as habitations for human beings. There are certain districts in London in which a second great fire is much to be desired, and on the areas thus cleared I would like to see dwellings erected each with its free hot- and cold-water service. For I fancy that few of us here would bath as often as we do now if only

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cold water was to be obtained on a chilly winter morning. The authority of sanitary science has been enormously extended in our time ; let us hope that we may live to see it able *to enforce the means of being clean.*

And as to niggardliness, I have often asked myself why it is that the public expect to pay less for a confinement than for the Christmas dinner, the bank holiday outing, or the baby's funeral, and I have come to the conclusion that it is mainly because the public regard labour as a healthy phenomenon comparable with the commoner natural acts of the body. Now whatever labour may have been in primæval woman it is certain that it is in most respects as real a disease as any you will find named in the official catalogue of the Royal College of Physicians. Nor does this statement apply to civilised races only, for amongst savages childbirth has its disasters and mortality, whilst deaths from this cause are common amongst domestic animals.

This belittlement of the importance of labour has reacted ill both on the patient and on the medical man. On the patient because it disinclines her to pay a fair price for the time and trouble that a confinement properly conducted entails, and on the doctor because he commonly earns no *kudos* for a cleverly managed case, and much obloquy if anything goes wrong. *The profession should sedulously impress upon the public that midwifery is a purely surgical art, and that as a surgical art with all its necessary accessories it should be adequately paid for.*

It is a ridiculous fact to consider that the average fee paid for removing tonsils is probably equal or greater than that commonly received for the conduct of labour, and that whilst in the first case an anæsthetist and all proper surgical accessories are allowed by the lay mind to be necessities for the success of the operation, the unfortunate accoucheur is expected to conduct *his* operation either without an anæsthetic at all, or by hovering about between the head and buttocks of the patient as he valiantly endeavours to combine the offices of anæsthetist and surgeon in one.

### The Duty of the Medical Man.

And in conclusion let us consider what steps we as medical men in the exercise of our profession may take to lessen puerperal morbidity.

*The use of gloves.*—First amongst these I would place the habitual use of india-rubber gloves in the practice of midwifery. It is less than ten years ago since I introduced them at the Chelsea Hospital for Women, and at that time comparatively few surgeons in England used them.

To-day it is an exception to see an operation performed without them, whilst even in midwifery, backward as it is as compared with other branches of surgery, their use is daily gaining ground. The use of gloves is beneficial not only to the patient being operated upon, but to all those subsequently to come under operation, because it protects the hands of the medical man from possible infection.

Further, when gloves are put on, as they should be, by previously filling them with some weak antiseptic solution, the hand is compelled to soak in that solution for a long time, instead of the casual one-minute "dip" which too often passes for efficient sterilisation.

The objection urged against them that they spoil the tactile sense is groundless. Far finer manipulations than are ever required in midwifery practice can be carried out in them as well as with the naked hand.

*The early treatment of abnormal labour.*—The duration of labour should be well under twenty-four hours in every case, in multiparæ much under. Directly it is obvious that this is not going to be the case interference on surgical lines should be undertaken, for there is a direct relation between the duration and morbidity of childbirth. Or, to put it in another way, the normal effect of uterine contractions is to dilate the cervix and to drive down the child. Were this fact, known of course to everyone, used as the sole criterion by which the proper action of the accoucheur is to be directed, we should not see these cases of prolonged labour, all too common, in which assistance to the obviously overburdened uterine muscle is obstinately withheld under the plea that it is our duty to leave the case to Nature to complete if she possibly can do so. Taking a case in the second stage, for example, it does not need two hours of senseless watching to ascertain that the power of the uterus is inadequate for its task. Given half a dozen strong pains and no advance of the head, and it follows from our criterion of normality that something is wrong, and that it is our duty to interfere.

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*On adequate surgical interference.*—Unnecessary meddling with a case of normal labour is, of course, to be condemned, but when it is necessary to interfere I would put in a strong plea that that interference should be thorough and surgical. One would laugh at the idea of endeavouring to anæsthetise a patient and perform an operation for, say, hæmorrhoids at the same time. Yet a parallel proceeding is constantly seen in obstetric practice. It should be postulated that the proper management of abnormal labour is impossible without the services of an anæsthetist, and the public should be made to understand that it is their duty to render this possible by paying an adequate fee for the proper conduct of labour. I hope to see the day when *two* medical men, the anæsthetist and the surgeon, will be held to be as necessary in abnormal childbirth as they are admitted to be in every operation of surgery other than the obstetrical.

*Never interfere unnecessarily, but having decided to do so in the patient's interests, do it like a surgeon.*

Thus, in a case in which the application of forceps is indicated, apply them under full anæsthesia, and before applying them make a thorough exploration of the head with the gloved hand. In regard to this I might mention that the position of the *child's ears* are the surest way to judge the lie of the head. The feeling of suture lines with one finger, as commonly practised, is fraught with mistakes, but you cannot conveniently feel the ears without an anæsthetic. At the same time that the lie of the head is ascertained the degree of tightness of fit between the head and the passage is ascertained. This preliminary examination will prevent such egregious errors as the application of forceps to the head in brow position or to the buttocks in mistake for the head. The manual rectification of occipito-posterior positions, also, is easy under full anæsthesia and should always be attempted before the forceps are applied. In short, it is a good general rule never to apply them (except perforce) to a head unless it is lying occipito-anterior or (in the case of a face presentation) mento-anterior, both of which lies are mechanically favourable to easy delivery. An exact knowledge of the relations existing between the head and the passage, such as examination under anæsthesia imparts, allows one to judge at once as to whether delivery can be safely effected without risk to the well-being of the mother or the life of the child, and renders impossible those dreadful cases in which one hears of the forceps being applied on and off throughout a whole night, or the bed and patient being bodily dragged round the room in the attempt to deliver her.

The child delivered and separated, place the patient, still anæsthetised, upon a bed bath in the dorsal position and thoroughly wash up the external parts with an antiseptic solution and ascertain the extent of any perinæal laceration. Instruct the nurse to prepare a hot intra-uterine douche (temp. 120° F.). By the time this is done some twenty minutes will have elapsed since the birth of the child. If the placenta has not come away by that time inject 15 minims of ergotin and immediately introduce the gloved hand into the uterus and remove it. That done the uterine cavity should be thoroughly irrigated out and a vigorous retraction obtained. The perinæum should now be sutured and the external parts once more thoroughly washed up. The binder and pad are now to be applied and the patient permitted to come round from the effects of the anæsthetic. I have done this operation a great many times and have never seen anything but perfect convalescence follow it.

This is *surgical* midwifery. Compare the following with it: The head refuses to come down; the attendant suspects a persistent occipito-posterior position but cannot be sure. The forceps are applied without anæsthesia and with ungloved hands and strong traction is applied. After half an hour's pulling they slip off and are reapplied.

At the end of another half hour, the patient being very restive and unmanageable, the head is suddenly born with a run—occipito-posterior—the perinæum being badly lacerated. A good deal of bleeding follows, and in the lateral posture it is impossible to be sure whether it comes from the uterus or perinæum. Endeavours to express the placenta are made, which is partially forced into the vagina but remains hung up by a tail of adherent chorion. The fingers are therefore introduced, and the mass is removed, the patient complaining bitterly the while. The perinæal wound is now sutured, but what with the inconvenient lateral position, the restiveness of the patient, and the continual oozing from the vagina,

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the sutures are badly placed and the job is only half done. This is *unsurgical* midwifery. Let me ask you which of these two patients is the more likely to give trouble in the puerperium?

Gentlemen, I fear I have over-taxed your patience. If so it is because I feel so strongly that the recognition of midwifery as a pure surgical art is urgently called for.

It is not the normal labour left entirely to Nature that commonly "goes septic," nor that in which artificial aid is given with all the accessories and precautions of modern surgery, but it is that in which a certain amount of interference is carelessly and unsurgically practised.

*August 17th*, 1908.

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**The Treatment of Erysipelas.**—D. B. Allen extols the use of carbolic acid in this disease, but claims that the strength of the applications commonly advised is far too weak to obtain satisfactory results. Proceeding on the theory that the affection is an infection of the subcutaneous tissue and lymphatics by the streptococci, he advises the painting of the area involved with the pure acid, extending the operation three eighths of an inch beyond the line of demarcation. This is allowed to remain until it becomes white, when it is washed off with 95 per cent. alcohol. The penetrating action of the carbolic is a direct germicide to the streptococci already present, and the ring around the area serves as a strong barrier beyond which it is almost impossible for the infection to spread. This line of treatment causes an intense smarting and burning, which lasts but a few moments and is quickly relieved by alcohol. In some cases the burning continues for an hour or two in which a cloth saturated with alcohol is laid on the area. In case of extension into the eyelids where it is impossible to use the pure carbolic, a 2 per cent. carbolic acid solution in unguentum hydrargyri ammoniatum to be applied frequently is ordered. The results in a series of twelve cases thus treated have been more satisfactory. The skin heals without scarring.—*Medical Record*, vol. lxxiv, No. 4.

# RHEUMATISM.

A discussion at the Hunterian Society.

**MR. ROWLAND HUMPHREYS in the Chair.**

Dr. Fredk. Taylor.
Dr. F. J. Poynton.
Dr. Horder.
Dr. Luff.
Dr. Fred. J. Smith.
Dr. Otto Grünbaum.
Dr. Seymour Taylor.
Dr. George Oliver.
Dr. Fortescue Fox.
Dr. Leonard Guthrie.
Dr. Arthur T. Davies.

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(*Continued from p.* 288.)

Dr. Luff spoke on the irregular manifestations of rheumatism and their treatment. He said: The few remarks I have to make do not deal in any sense with acute rheumatism, or rheumatic fever as it is commonly called. No one recognises more than I do the authority with which the opener of any discussion should be invested, especially when that opener is one whom I respect so much as I do Dr. Frederick Taylor. But in limiting his opening, as he did, to acute rheumatism or rheumatic fever, I feel that I am to some extent placed out of court in the remarks I shall have to make. I see, however, from the card that this is a discussion on "rheumatism." Well, it may be taken that by that we are only to consider acute rheumatism or rheumatic fever. But in my opinion if it is to be a discussion on rheumatism it must be a discussion on those conditions which, rightly or wrongly—wrongly as I think—are called rheumatism, are recognised by the public as rheumatism, are recognised by members of our profession as "rheumatic" at all events, and which are so constantly demanding our attention. I think I am not exaggerating when I say that as practitioners, where we have to treat one case of acute rheumatism or rheumatic fever we have to treat something like 100 cases of so-called rheumatism. It is to these cases of so-called rheumatism that I am going to briefly refer. The term, of course, is an unfortunate one. It has cropped up mainly because, as regards the pains, and as regards the parts which are so painfully affected, they are the parts which are similarly affected in the majority of the cases of acute rheumatism. I am referring to the subject of so-called chronic rheumatism. By that I mean that pain which is produced in the fibrous tissues in the various parts of the body. It is nothing

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whatever to do, pathologically, with acute rheumatism; it is a chronic inflammatory hyperplasia of the white fibrous tissue in various parts of the body, sometimes the white fibrous tissue in the joints, sometimes the fibrous tissue in the muscles, sometimes the subcutaneous tissues, sometimes the aponeuroses and tendons and tendon sheaths and periosteum. This condition constitutes what is sometimes called muscular rheumatism, or sometimes fibrous rheumatism. As to the causation of this so-called chronic rheumatism, in the first place I think it would be well if we were to drop the term "rheumatism" altogether in connection with it, and in its place to use the term which I think was first coined by Sir William Gowers, namely fibrositis, that is to say an inflammatory affection of the fibrous tissues. And if you will permit me, sir, in my few remarks, I shall refer to the condition as fibrositis, and not as rheumatism. It is not started by whatever may be the exciting cause of acute rheumatism. And if I may briefly refer to its origin I would divide it into three main causes. In the first place, this fibrositis, or so-called chronic rheumatism, is frequently started, as we know, by exposure to wet or cold. As examples of that there are lumbago, intercostal rheumatism, stiff neck, and the various forms of so-called muscular rheumatism. It is a common experience that this inflammatory hyperplasia of fibrous tissue is started by exposure to wet or cold. This fibrositis may be started by strain of some sort, the so-called muscular rheumatism of athletes, that which is produced by various games, particularly the "tennis elbow," the "golf elbow," etc., or it follows the prolonged strain, if I may speak of it—and I think I may—which results in that peculiar chronic fibrositis know as Dupuytren's contraction of the palmar fascia. In all these cases of fibrositis which are induced by strain, when once a part is affected it often is not completely cured, for the slightest strain or wrench afterwards may bring the pain on again. It is a common experience for pain to return in the part when it is subjected to slight strain or extra pressure. A third way in which this fibrositis is produced is an important one to remember, because it may bring us into touch with the pathology of rheumatic fever, and that is that it may be produced by the irritation caused by various toxins, generally by toxins absorbed from the alimentary tract. As illustrations, there are those peculiar muscular pains which many people experience after excessive indulgence in rich food and a variety of wines. Those pains are too frequently ascribed to gout, but as a matter of fact they have nothing whatever to do with gout. The pains which affect the various parts of the body after indulgence in rich dinners and various wines are due to the absorption of irritating toxins from the intestines. As further illustrating this there are the pains which follow tonsillitis, the pains which follow influenza and which I believe to be due to irritating toxins, and those which follow a febricula or common cold. In all these cases of fibrositis there is this induration of fibrous tissue, and in them a fibrous mass can be felt in the subcutaneous tissues, and frequently in the joints and in the muscles and in the periosteum. Any compression or stretching of these indurated masses produces very great pain.

I now propose to approach what has not been approached in this debate, with the exception of the reference to salicylates, and that is the question of treatment. After all that is one of the most important matters which we have to discuss. As the time is late I shall only refer to two of the commonest forms of what I should call fibrositis, because they both well illustrate the special treatment which should be adopted. I refer to what is commonly called lumbago, and that common affection of the knees which is sometimes called chronic rheumatism, and frequently, though wrongly, called rheumatoid arthritis, but which I prefer to call a chronic villous synovitis in which there is creaking and pain and fibrositis affecting the joint. First, then, with regard to lumbago, I shall take as representative of that the involvement of one or other of the sacro-iliac joints and the fibrous tissue around them. In the majority of cases of lumbago pain is complained of in the lower part of the back. The lumbar muscles are not involved at all. You can examine the quadratus lumborum and it is not tender, but if you make pressure over the sacro-iliac joint the patient will complain of pain. That is the real seat of the fibrositis, the attachment of the tendons in the vicinity of the sacro-iliac joint, and that is why such lumbago is followed by sciatica, and that inflammation spreads through and involves the plexus in front of the joint. As to the treatment of lumbago,

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in the first place salicylates and the salicyl derivatives are useless except as relievers of pain in the treatment of all forms of fibrositis. As pain-killers in acute lumbago I know of nothing better than a combination of aspirin and pyramidon, the latter being an amido-derivative of antipyrin, which has not the depressing effect of antipyrin, but relieves the pain equally well. But its action is simply to relieve the pain, because as soon as you leave off the remedy the pain returns, and that of course is not attacking the actual disease. In attacking the disease itself, there is only one drug in my own opinion—though perhaps this statement will be questioned, and I hope it will—which, given internally, attacks this fibrositis or chronic rheumatism, and that is potassium iodide. I give it in the largest possible doses. With regard to the local treatment in these cases of lumbago, there is no doubt as to the way in which it can be relieved. There are two ways especially in which this local fibrositis may be relieved; one is the application of heat in some form, and the other is ionic medication, in which a drug is driven in by the continuous electrical current. The drug may be iodide of potassium, which is introduced by cataphoresis, or it may be iodine and carbonate of lithia, manufacturing iodide of lithia on the spot. What I generally direct in all cases of lumbago is that the patient should have an electric light bath for a quarter of an hour, with special concentration of the rays on the painful area, and after the douching which should always follow the electric light bath, iodine liniment is painted over the painful area, and a lint pad saturated in a solution of carbonate of lithia laid on. The continuous current of 7 to 12 milliampères is then passed, the positive pole being on the lint pad, and the negative pole on the thigh lower down. In that way in ten minutes all the iodine which has been painted on the skin disappears, and ionic medication of the affected fibrous tissue has been brought about. In my opinion that method of local treatment is the best for relieving the pain of lumbago, which may be very severe. What I have said applies to the treatment of chronic disease of the knee-joint, to which I referred as chronic villous synovitis. In my experience there is no necessity to diet patients who are suffering from this chronic condition. The idea that it is connected with gout is erroneous; the idea that these conditions of fibrous tissue in the joints are connected with uric acid is absurd in my opinion. I hope we in the medical profession are about to abandon once for all this absurd fetish worship of uric acid. Uric acid is only a harmless by-product, the *débris* of various processes, and it has nothing to do with the pathology of either gout or the various forms of rheumatism which we are considering.

Dr. F. J. SMITH: The subject of rheumatism must interest us all, because what the public call rheumatism is exceedingly common, and I take it that the only notion which the public has about rheumatism is that it is anything which is painful and especially anything painful in the limb. And that, of course, gives point to Dr. Luff's remarks about the better term "fibrositis." There has been such a very rich feast of fact, fancy, and theory put before us to-night that it is exceedingly difficult to pick out a bit and really elucidate it. But it is my usual *rôle* to play the part of a friendly critic, and I would like those who have used the term "malignant endocarditis" to define what they mean by it. We must all recognise that between the very simple swelling of a valve of the heart which probably occurs in acute rheumatism, and the valve which is absolutely destroyed by ulceration, there is every conceivable intermediate grade. And what I am accustomed to tell the students at the hospital is that every case of endocarditis is ulcerative. It must be, in the very nature of things. The endothelium comes off from a swollen valve, and to use the term "malignant" for any form of it is, I think, wrong. I was going to say it is wicked, and I think it is wicked in the mouths of scientists; they have no business to use it. If they want to speak of endocarditis which kills and is due to a toxin, let them tell us so in plain language, and if it is possible to do so let them also tell us what microbe it is that is doing it. I have been told by bacteriologists that every microbe yet known to science has been found on the valves of the heart, and in that case they should drop the term "malignant endocarditis" absolutely, as well as the term "ulcerative endocarditis." The term "endocarditis" should be used simply, adding an adjective to indicate what particular microbe is at work causing it. If they accept this diplococcus as the cause of rheumatism, let them use the term "endocarditis diplococcia," or

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"endocarditis rheumatica" if they prefer that term. If it is due to the gonococcus, or to the pneumococcus, or to the diphtheria bacillus, let them say so. Let us have plain speaking, and not words which mean one thing to one man and a totally different thing to another. So much for the terms used to which I object. The points which Dr. Taylor has raised seem to me to be of great interest, and I have learnt things to-night touching chorea which I suppose have been more or less simmering in one's brain, but upon which I have found it extremely difficult to teach students, because one has felt hitherto so particularly ignorant of its meaning and its pathology. But Dr. Frederick Taylor threw a flood of light into my mind when he mentioned it in connection and comparison with tetanus and hydrophobia. We think we know something about hydrophobia, and we think we know something about tetanus. We think tetanus is a pure chemical toxæmia, that no microbes get into the blood. And that gives one an exceedingly clear notion of chorea and its meaning, that it is a pure chemical toxæmia. I do not think I can add anything that is really useful to this discussion, and I will content myself with thanking the speakers who have so ably introduced it, especially Dr. Frederick Taylor.

Dr. Otto Grünbaum: Sir, I came because I thought I should learn much about this disease, and I have been far from disappointed, because I have learnt a great deal, and I hope that by asking a few questions I may learn still more. Some years ago I attempted to grow cultures from the blood of cases suffering from what I believed was acute rheumatism, but in every case I failed. Therefore I should like to ask Dr. Poynton in what percentage of cases examined cultures have been obtained, and how many of these have been ante-mortem examinations, that is to say some ten days or more before the decease of the patient. That of course is an extremely important matter, because a terminal invasion occurs in so many diseases. There are many arguments which could be raised against the micro-organism being specific, and Dr. Poynton told us he was aware of most of them, and would be glad to point out that most of the arguments could be met by counter-arguments. The chief experiments I am acquainted with which tend to point towards the micro-organism isolated by Poynton and Paine being specific were those showing that formic acid is manufactured, and also that if this micro-organism is grown in broth for a long time and the broth filtered, it is possible to make other streptococci grow in this exhausted broth while the *Diplococcus rheumaticus* will not grow. But of course that is not absolute evidence of the micro-organism being specific. In considering the huge number of streptococci which exist, it seems to me that we want evidence beyond this that the *Diplococcus rheumaticus* is something different from the micro-organisms which have been described by Drs. Andrews and Horder. If I could obtain evidence that the diplococcus has been grown from a considerable percentage of cases that have recovered, I shall feel that the evidence of this diplococcus being specific is greater than I anticipated.

Dr. Seymour Taylor: I had intended to make a few clinical remarks about the condition of rheumatism in children. I have been teaching for a number of years certain doctrines, which may be wrong but which I think are correct, and I believe they have never been clinically disproved. I contend that acute rheumatism, which is what I am really speaking of, is a specific disease and has many manifestations. In one man it will show itself entirely as an endocarditis, or pericarditis, or myocarditis, and in another person it may be a muscular affection, and in a third person the disease is spent on the joint. Another patient comes under treatment for all these conditions. Sometimes, though admittedly very rarely, you see rheumatism in the form of iritis, or in the shape of rheumatic nodules. These are well-known clinical facts. But I have also taught, and it was one of my first experiences as an apprentice, that tonsillitis is a form of rheumatism. I am not quite sure of my ground here. As a result of experiments made chiefly in the laboratory, I think that the condition of the tonsil being unhealthy serves as a port of entry rather than as a manifestation of the disease. But there is one aspect of the disease which is extremely important, namely, its bearing upon child exposure. I have said for a number of years—though it was not an original observation on my own part, but I have insisted upon it more strenuously than others—that chorea in the child is rheumatism. I am not speaking of the different forms of chorea

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in the adult. I refer especially to that condition in the child from three or four to twelve or fifteen years of age. In proof of that I have taken out some figures, and the majority of them showed to me these certain facts, viz. that with the choreic child one or other parent is rheumatic; that the disease is preceded by, or complicates, an attack of rheumatism in a large number of cases, that it runs therefore in families and in those with a rheumatic tendency. In severe cases of chorea there is a rise of temperature observed frequently, and one more important fact is that in 30 per cent. of these children there is pericarditis or endocarditis, or at any rate they have heart disease, just as do a certain percentage of cases of acute rheumatism so-called. Another important point about this relation of chorea to rheumatism, although I call it the same disease, is the fact that chorea yields to treatment by salicylates sooner than to arsenic or to other vaunted remedies. I should like to tell you of an instance in which I was corrected by my house-physician. We had in the hospital a young child, æt. 14 years, so acutely affected with chorea that we had to have ropes round the bed to prevent the severe jactitations precipitating the child out of bed. And in speaking to the post-graduate class we discussed this possible relation of chorea with rheumatic fever, and the history of rheumatism. We got from the father and mother an indignant denial of rheumatism. My house-physician said: "Why not try salicylic acid treatment?" The reply was that there was no history of acute rheumatism, and therefore it would be probably useless. The child was put on gradually increased doses of Fowler's solution, and even poisonous doses, but without relief. One afternoon I could not get to my ward, and the next time I went round I saw the patient and at once said, "The arsenic has done her good." My house-physician at once said, "She has had no arsenic since your last visit, and I put her upon salicylate of soda." That was a great lesson to me. When a mother in the out-patient room tells you that "the child had rheumatic fever and it went to the brain," and that "afterwards the child had St. Vitus's dance," she speaks correct pathology. I have had a child in the hospital in connection with whom there was no history of rheumatic fever, but who suffered from chorea. We did a lumbar puncture, and the micro organism of Poynton and Paine—for I think it should bear the names of our distinguished colleagues—was demonstrated. That patient was cured quickly by the administration of salicylate of soda. My remarks are very disjointed because I wanted merely to give you the gist of my experience. I cannot leave this meeting without asking you and asking my colleagues here to believe with me the statement which I make (and which has been inferred by other and better men than myself, such as Sir Dyce Duckworth, Dr. Garrod, and Dr. Herringham in this country) that chorea in a child is another name for rheumatism. I do not think any of those physicians have gone so far as I have, namely, to say that chorea and rheumatism are one and the same disease, which should be treated by the same remedies which we apply to acute rheumatism, but it is a statement which in my experience is capable of clinical and pathological proof.

Dr. POYNTON, in reply, said: I would apologise to Dr. Horder if he thinks I am too controversial: perhaps it is due to my having come from a wrestling county in England. As to the remarks about terminal infection, I should like any such idea to be done away with, and I really think we can put it aside. When one finds this organism people say it is so easy to say it is a terminal infection. I can vouch for a patient living and working six years afterwards in whom we got the micrococcus out of the blood during acute pericarditis. I know of another case of arthritis which certainly has left the hospital, and a case of chorea in which one got it from the cerebro-spinal fluid. And there is the convincing case of Dr. Lee's, which lived a month after isolating the diplococcus from the blood. The patient died, and one was able to prove that the case was one of rheumatism. There is yet another case in which Dr. Thiele isolated it from the blood at University College Hospital. I think, therefore, we may put aside this question of terminal infection. I have never been able to follow Dr. Horder and Dr. Andrews with regard to *the Streptococcus fæcalis*, because the fæces are not by any means a constant medium. We do not know what changes these streptococci may undergo in a medium such as fæces. Such a fact as the isolation of a micrococcus from a cardinal lesion of rheumatism is worth, in my opinion, a thousand observations on the fæces,

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where there are sure to be all sorts of organisms, including that of rheumatism. With regard to simple and malignant endocarditis, I am glad to hear Dr. Smith say that there is no real distinction between them; you get every gradation but it is the same condition, and that is true in animals and in man. One animal here had simple endocarditis for months, and died later of malignant endocarditis. So anything which may attack the valves, be it rheumatic, gonococcal, influenzal, may produce simple endocarditis and malignant endocarditis. With regard to a specific micro-organism, so many bacteriologists speak of this specific micro-organism as if, could you only magnify it enough, you would be sure to see the word "rheumatism" written on its back. These organisms are not so specific. We have never claimed we could get a specific test for this organism. We have said in regard to it that we believe it to be the cause of rheumatism because it is in the lesions of rheumatism and can be got from them, and having been obtained we can produce similar lesions in animals by injecting it into them. There are certain minor distinctions which have to be pointed out, which Dr. Beattie pointed out, but these are minor distinctions as I say, and we have always to remember that this micrococcal group is a very difficult one.

Dr. F. J. SMITH: There is one question I want to ask. Have Drs. Poynton and Paine tried putting salicylate into one of these growths to see if it would check it? Is there anything specific about it in that way?

Dr. POYNTON: We have tried that and we have tried salicylates with rabbits too. But salicylates check growths so quickly that it is difficult to argue from the test tubes. It is antiseptic and therefore will check many different kinds of growths.

(*To be concluded.*)

**Anti-gonococcic Serum.**—A. A. Uhle and W. H. Mackinney report results of the use of anti-gonococcic serum in twenty-three cases of the various types of gonorrhœal infection. They find that, so far as their observations indicate, the serum has little if any curative action on the urethral condition, the improvement observed in some patients being more attributable to rest and hygienic treatment than to any specific action of the serum.—*Medical Record*, vol. lxxiv, No. 4.

# THE SURGICAL TREATMENT OF THE IMPERFECTLY DESCENDED TESTICLE.*

By EDRED M. CORNER, M.S., F.R.C,S.,
Surgeon to St. Thomas's Hospital and the Hospital for Children, Great Ormond Street, in charge of Out-Patients, etc.

IT is impossible to state hard and fast rules of treatment for cases of imperfectly descended testicle, because they vary so much in their imperfections of descent and also in their concurrent deficiencies, imperfections of development. These are two points of greater magnitude to bear in mind in treating these cases, the imperfect descent and the imperfect development. To improve the imperfect descent by operation in reality does the patient very little good. Indeed, although much can be done by operation to cure the imperfect descent, unfortunately little can be done to improve the second factor of imperfect development. It is impossible for us to make a dwarf grow to a Sandow or an imbecile become a Darwin. They have not the requisite capital. Similarly, we cannot make a testicle grow to full size and maturity if it is not so designed and capitalised. In consequence, operations which are merely aimed at curing the imperfect descent of an imperfectly descended testicle must fall into disrepute because they do not improve the development of the gland, which is by far the more important factor. Thus by neglecting the question of the future development of the gland the results of operations of orchidopexy are unsatisfactory. But not only do the operations of orchidopexy not improve the development of the imperfectly descended testicle, but often they actually do harm by causing venous congestion and inflammation in the gland. Tersely it may be stated that orchidopexy is an operation which fails very often both anatomically and physiologically—anatomically, because the gland fixed in the scrotum seldom remains there; physiologically, because the testicle is largely converted by it into useless connective tissue instead of useful epithelium. Except in very carefully selected cases it is commonly a dangerous operation. But as it has been hitherto

* Part of a discussion at the Harveian Society.

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the most popular operation for imperfect descent of the testicle, it is necessary, having condemned it so heartily, to examine very carefully into the other lines of treatment left to us, which are three.

The first, to leave the case alone:

This line of treatment can be pursued safely until one or any of the following occur: (*a*) attacks of pain in the testicle; (*b*) attacks of inflammation in the testicle; (*c*) hardening of the testicle; (*d*) dilation of the inguinal canal or the development of a hernia.

The second, orchidectomy or castration:

This is only justifiable when the spermatic cord is twisted, the testicle much sclerosed or the patient twenty-three years of age.

The third, orchido-cœlioplasty, or the abdominal reposition:

This is the most useful operation from the patient's point of view, because it neglects the comparatively unimportant imperfect descent of the gland and ministers to the all-important imperfect development of the gland. An imperfectly descended testicle can rarely, if ever, form spermatozoa, that is to say, become spermatogenetic. So that its whole value may be taken to lie in its internal secretion, which is fostered by the abdominal reposition of the gland, and not in its external secretion.

The reposition can be done in two ways—an extra-peritoneal orchido-cœlioplasty and an intra-peritoneal orchido-cœlioplasty. The latter is preferable, the gland being placed under conditions similar to those of the ovary. Increased liability of the onset of malignant disease is not incurred after orchido-cœlioplasty, because it is not the abdominal position of the organ but the concurrent imperfect development which gives it its additional proclivity to become malignant.

From what has been said, particularly the four factors which should terminate the period of treatment by waiting—pain, inflammation, hardening, and dilation of the inguinal canal, it will be seen that very few cases of imperfectly descended testicle will escape operation, especially when it is remembered that quite 80 per cent. are associated with a hernia sac. And having explained my ideas on the scope and limitations of the various lines of treatment it only remains to state the cases in which the operation of orchidopexy is useful. It is particularly indicated in cases of movable testicle, slight imperfections of descent, and in the younger subjects, say under seven years of age.

*August 17th*, 1908.

## CHRONIC NEPHRITIS.

EMERSON records a series of careful experiments on fourteen dogs, extending over twenty-six months. From experimental results it would appear that interference with the blood supply of the kidney can be accomplished by means of chemical injury to the renal parenchyma, and that the process of repair which ensues may involve such a destruction of secreting tissue as to cause the death of the animal. From the experimental use of general vaso-dilators it appears that the kidney suffers at least mild grades of degeneration, which may be due, not to the specific renal irritation of the drugs employed, but to the defective blood supply which frequently repeated vascular relaxation causes. From an analysis of the accepted causes of chronic nephritis in man it is evident that many of them have, as a common factor, a more or less severe and prolonged stagnation in renal blood supply, and, although this factor is often accompanied by the presence of irritants which we recognise as injurious to the kidney tissue, insufficient blood supply alone will cause, first, an error in the function, and later, an alteration in the structure of the kidney. Loeb's explanation of the probable sequence of the circulatory events coincident with the course of chronic nephritis in man might reasonably be supplemented by an explanation of the ætiology of nephritis, which recognises as a frequent, if not an invariable factor, deficient renal circulation. Faulty function always results from deficiencies of blood supply, and if the defects persist or are frequently repeated faulty structure results. In the case of the kidney, the fault in its function resulting from the altered structure in chronic nephritis will cause much or little disturbance in the body at large, according to the degree of circulatory compensation which can be maintained. Cardiac disease, bacterial toxins, and metabolic waste products are probably the causes of the errors in circulation which cause, first, faulty function in the kidney, and, finally, chronic nephritis. If this theory of ætiology is supported by subsequent study, its application to the prevention of nephritis might strengthen existing efforts to check the spread of infectious disease, and to urge moderation in the habits of over-stimulation and overwork, which are prominent in present-day life in large centres of population.—*Journ. A. M. A.*, vol. li, No. 4.

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# MENTAL DISTURBANCE IN WOMEN.

A Clinical Lecture delivered at St. Bartholomew's Hospital.


By NORMAN MOORE, M.D., F.R.C.P.,
Physician to the Hospital.


GENTLEMEN,—I propose to-day to lecture on a certain series of varieties of mental state in women, which we have lately had the opportunity of seeing in the wards, and about which it is important you should know something. Cases of affection of the mind come into the practice of every physician, and their consideration is an important part of his work. You are sure to come across plenty of examples of mental disorder, and it is important you should understand them. The first example I want to bring before you is that of a woman in Hope Ward, æt. 26 years. She was admitted during lactation. When she came in she had a child about ten days old, and her condition I will describe to you. The child was her third, and she was taken ill when it was about eight days old, her chief symptom being shortness of breath. She brought her child into the hospital with her. On March 20th she had a severe pain in her left side. On March 23rd there was more pain there, and on March 24th dulness and absence of breathing were discovered at the left base. Notice the gradual onset of the condition. On March 25th, the day of her admission, she had impairment of resonance varying in degree over the whole left back, and absence of breathing. Her temperature was 103°F. It had been raised from the beginning of her illness. On March 26th she had bronchial breathing on the left side. On the next three days there was some impairment of resonance, and some loss of breath-sounds on the right side. When you hear those notes and look at the temperature chart you will perhaps incline to the view that her condition was one of pleuro-pneumonia; the temperature was

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high enough for it, and the day on which her temperature fell corresponded with the common course of that disease. The only point on which there was difficulty in believing it was a continuous case of pneumonia, in which one lung was affected first and the other one was infected from it, was that after the fall there was not any particular rise. However, I think there can be no doubt that she had pleuro-pneumonia, and that her left lung was the one chiefly affected. I think that she afterwards had some slight infection of the lowest part of the right lung. She was admitted on the 25th, and on the 26th the baby was admitted, and the baby was kept in a basket by the fire in the ward. On the 26th she was nursing it. Her mental state at this time was obviously not clear; she did not answer questions readily, and she was apparently very unwilling to answer them at all. On the 27th, 28th and 29th her mental state continued somewhat obscure. But you know a person with pneumonia and such a high temperature might easily fail to answer questions very readily. Her mental condition, however, was not like the delirium of pneumonia; it was mental obscurity rather than active delirium. On the 30th she tried to get up, and made an attempt to bite the nurse, and seemed disordered in her mind. Her ideas were obviously disconnected. The following morning she was rational and answered questions. All that day she sat up close to the edge of the bed, with a very anxious look towards the child. Lactation had to be put an end to and the child sent home. As she sat there the mother had a look of extreme anxiety, and in the intervals of anxiety she seemed suspicious, and was constantly looking, after the removal of the child, to where it had been. She reminded me of the following passage, which I want to read to you, because I wish to impress upon you that though this woman was undoubtedly for the time being out of her mind, it was a condition of mental disorder closely allied to a natural condition, but exaggerated. I do not know if you have ever read the story called 'The Hoggarty Diamond.' It is one of Thackeray's earliest stories. In this story the wife of the man about whom the story is told loses a child, and they are in very distressed circumstances, in fact, the husband is in prison for debt, and his wife decides to go out as a wet nurse. She goes to the house of a great lady, and there is waiting with other candidates to see whether she will do for the post. A nurse came out of the room with the lady, "and," says the reporter of the scene, "while the lady was talking to us she was walking up and down in the next room with something in her arms. First the lady spoke to Mrs. Horner, and then to Mrs. Titmarsh, and all the while she was talking, Mrs. Titmarsh, rather rudely as I thought, was looking into the next room, looking at the baby there with all her might. My lady asked her name, and if she had any character. And as she did not speak I spoke up for her and said what was to be said. 'Poor thing,' said the lady, taking Mrs. Titmarsh's hand very kindly, 'she seems very young.' 'How old are you?' 'Five weeks and two days,' said your wife sobbing. Mrs. Horner broke into a laugh. There was a tear in my lady's eyes. At this moment the child in the next room began crying. As soon as your wife heard the noise she sprang from her chair and made a step forward, and put both hands to her breast and said 'Give it me,' and began to cry again." That little account I have no doubt is true to life. Many of the circumstances in this story are known to be taken from incidents in Thackeray's own life very early in his career, and he described what he had seen. It is admirable. This poor young woman was so absorbed in thinking of her child that she mentioned its age as if it were her own, and her own mind is turned with sympathy towards the child that is to be nursed. This woman in the ward constantly looked towards the basket, and after the child was removed thence she continued to look at the place were it was, as if she was altogether unable to take her mind from the subject. And although we assured her that the child was going on well at home, that did not satisfy her. She still constantly had this anxious appearance, and was clearly in a state of ephemeral transitory mental disturbance; I hardly like to call it mania, though it is one of those conditions which would have been included years ago under the general term "puerperal mania." But it is not strictly a diseased condition of mind. It is only carrying natural maternal anxiety a little further than is necessary. I dwell on it to you because I want you to know that when you see a case like that you may feel sure that the patient will get well, that she will gradually recover. I was obliged to send this woman to a separate

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room in Casualty Ward, as it was difficult to look after her in the ordinary ward. There she gradually came round and got quite well. But as I talked to her just before she went I could see that she had only just got rid of the suspicion that something wrong was going on with regard to her child, or that the child was not being properly looked after. She knew the suspicion was unreasonable, but her mind was in that state that it was only by a very great effort that she could satisfy herself of all being right. Directly she gave up that effort she sank back into the state of partial mental disturbance and again thought that all was not right. That anxiety could be seen perfectly well in her face. It is a very important thing to bear in mind in medicine that there are physical conditions in women which, although such patients are conscious of their physical conditions, seem to get to a very slight degree beyond their control. Of course one large group of these conditions is that which we do not often see in the wards, but of which you are sure to meet with innumerable examples in private practice—the mental disturbance which occurs at the menopause. It takes an enormous variety of forms. It is most important when you are consulted about the health of a woman between forty-two and fifty-three to remember that that element should come into your consideration. Very often the mental state takes the form of anxiety lest the patient should have some disease which relatives have had, particularly cancer. Women at that period of life who have no real indication of cancer in any part of the body, and no reason to think that they are likely to have it, are nevertheless filled with apprehension about themselves on the subject, and sometimes such women feel that they will go out of their minds; they think there must be some remote history of insanity in the family. Probably they are reasoning sensible women, and would say to themselves, "This is unreasonable and I need not bother myself about it"; but there is something in the condition of the menopause which causes the mind to slip from under their control as it were, and they cannot act on themselves with that force of character which at another time would be quite natural. Clearly you will see how important it is in dealing with them to remember that what you have to do is to try to put anything which you say about their case into such a form as shall absolutely convince them. It is not impossible; it can be done. By examining carefully and thoroughly you can demonstrate to them, although they are without medical education, that they have not a new growth, or tuberculosis, and in the same way by reasoning the matter out you can demonstrate to them that they have no real sign of insanity—I press that upon you. You cannot imagine how many cases of that kind you will see in practice, and how firmly you must make up your mind about them if you are to be of any use to the patient. Of course it follows that you must first convince yourself thoroughly that there is nothing wrong. There is another condition of transient mental disturbance in women which I consider belongs to the same group of conditions, in which the physical condition causes the patient's mind to slip, as it were, beyond her control. That is the condition which occurs in a very large proportion of women at the menstrual period, not merely when the menstrual period is disturbed, although it is more common in cases where menorrhagia may be described as present. But even in cases where the menstrual period is quite natural it may be present. But in most of the cases of the kind there is some excess of the period present. The mental disturbance may be very serious indeed, and the patient is conscious of it, but yet may be a very reasonable and intelligent person, and it is only with the greatest effort that she can prevent her mind slipping from her own control. The signs of mental disturbance are very variable; there may be excessive bad temper, and extreme suspicion. Not long ago I was consulted by a lady of considerable wealth; her husband had not very much wealth. They had a grown-up family, and at the menstrual period she was always seized with peculiar and quite unjust suspicions that her husband was likely to spend a great deal more than he ought to spend. She herself had the feeling that there was no ground for the suspicion, but nevertheless it made the household thoroughly uncomfortable. That poor woman in the ward perhaps was hardly conscious that her mind had got beyond her power of control, but it had completely. For many hours in the day people at the menopause often feel apprehensions which they know are foolish; these and the patients who have disturbance at the catamenial period may be

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regarded as a single group. I want to impress upon you that those conditions are distinct from what we call hysteria. Hysteria, as we see it in the wards, and as you will see it in practice, is quite a different thing; it is not due to a perfectly sound mind slipping away from control, it is quite different from that. I do not propose to lecture to-day on hysteria; it is a very complex subject. I will only say with regard to hysteria now, that hysteria occurs in the same characters in both sexes, and if I were asked to draw a line between hysteria—(Sydenham made what was a shrewd remark for his time, that hysteria occurs in the same form in male and female patients)—and the other states I have described, I should say that the states I have been describing have something to do with a definite physical state, either uterine or general, and that hysteria is not associated with any obvious physical condition. Many people only think of hysteria in relation to the uterine and generative system, because of the word, but it really has nothing to do with it. In the vast number of cases you will be able to notice that hysteria does not arise in that way. Hysteria must be due to some changes, but by our present methods they are not discoverable. But do not think of hysteria as a disease of sex.

I want to speak of one or two other conditions of mental disturbance, and I have confined my remarks to women because the cases upon which I am lecturing have occurred in women. The first three varieties are, of course, purely female. Next I want to speak about a mental state or disturbance following the administration of chloroform. That very likely occurs also in men, though, in a not very large experience, the best marked cases of it which I have seen have been in women. I will read you a few notes of such a case. The patients have had chloroform, or chloroform and ether, but I am unable to associate the state with any particular anæsthetic. The only essential circumstance which must be present to produce it is prolonged administration, such as for a long operation. After such an operation some patients are found to be nearly insane; it is most alarming. Here was a patient who had to undergo a prolonged operation on the mouth and tongue for the removal of a new growth of the tongue. Immediately after the operation the patient came to and seemed all right. She did not speak, but was thought to be weak, and wanted to get up. She was, of course, advised to stay in bed. I saw her on that day, which was four days after the operation. She pointed at things and muttered, but could not answer any question. She seemed utterly confused, but not unconscious. She was about forty-four years of age. Two days afterwards she wrote on a piece of paper the name of her house in the country, and the word "Queen." She had no paralysis. The movements of her eyes were perfectly natural; no sign of brain disease was present. On getting her out of bed she was able to walk with the help of two nurses. Two days later I found her crying very much, and unable to answer questions. I talked to her and tried to cheer her up, and I may say you will find it very important indeed to take all the pains you can in cheering patients up. I often think of a sentence in Petronius Arbiter, which is intended to be against physicians. He said: "Medicus nihil est nisi animi consolatio." I think it a great deal if the physician can be that. If the physician can cheer a patient he is very far advanced in his profession. I tried to console her, and she wrote on a bit of paper the word "Thanks." Later she wrote "Worse." She then spoke, saying "Black pencil." The pencil she had would not write very well. She seemed unable to say any more; she could not continue the expression of thought. She had a good deal of sleep, was very anxious to get up, and four days later she wrote, "I am going to London on Saturday night." She was in London. I said, "You are in London," and she shook her head incredulously. She wrote, "I want to see my mother." I said, "I thought your mother was not living." She replied, "Yes, she is." So she wrote in a partially rational state. The same evening she wrote on paper and handed it to me, "You know in helping me the nurse broke the tinkels." The thermometer had been broken, and that was her attempt at expressing it. I mention this to convey to you what was the exact disturbance of her mind. As one talked to her one might easily think she was out of her mind. She got up that evening and, taking the nurse's arm, walked round the room. She was a well-educated woman. I said, "You will remember the 'Voyage au tour de ma Chambre?'" She said, "Yes." We looked at the pictures in the room, and when we came to one showing the castle of Chilon she recognised it. She was shown a miniature of herself and recog-

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nised it, and also one of her son. And so she went on until four days more—that made altogether ten days. On the tenth day she wrote on a piece of paper, "Am much cheered by what you said." I asked her to name the artists who painted the portraits in the room, and she wrote down their names. Three days later her condition was quite natural. Of course, she was very ill. So that her mental disturbance lasted about a fortnight from the operation. It was not developed for the first few days. But she lay absolutely torpid. She did not live longer than about two months more, but she remained clear in her mind and talked rationally during the remaining part of her life. That was a very good example of mental disturbance coming on after prolonged administration of an anæsthetic. In the first instance I was asked to see the case to say whether she was insane or not. I said she was not insane and that she would recover. That was based on the fact that all the patients I had seen in that state recovered their normal mental state.

Now about the girl who is here. I want you to observe that she looks pale, as if she had been undergoing a very serious illness. She is not paralysed, she can move her limbs, and she does excellent needlework in the wards. There is nothing whatever abnormal about her nervous system at the present moment, but she is not strong enough to stand. We will send her back to the ward and discuss her state. She was in a state of mental disturbance when she came into the hospital. I will read you the note. She was admitted to the hospital on February 29th. On February 28th last year she and other members of the family got a severe sore throat; she was sent to bed and had an injection of antitoxin. From that it is fairly clear that the condition was thought to be diphtheria. On December 24th she was found in bed in a seemingly unconscious state, and did not recognise her mother. She persisted in that condition for some days and after a time refused all food. She was at times caught out of bed, although supposed to be paralysed on the right side. She got extremely emaciated. A nurse was obtained and fed her through a nasal tube. During that time her right arm and leg were kept fixed in an extended position, and the hand was tightly clenched. She either would not or could not talk at all, was excessively emaciated and gloomy, and she could do nothing with her right arm and leg. Her eyes reacted to light and to accommodation. When anyone came near her she seemed unable to say anything beyond "No, no." When one tried to examine her eyes she was quiet. When inspected from a distance she was found to be looking about quite naturally. There was nothing apparently abnormal in her physical state otherwise. What was the condition? She had no true paralysis, and she gradually recovered the use of her limbs. She was fed, and treated with electricity. Except for her excessive weakness she is now better, and takes her food well and talks well. I think it was primarily a case of mental disturbance, the toxic result of diphtheria. True paralysis was not produced, but a very great disturbance of the nervous system ensued, and the chief manifestation of that was the refusal to speak or to eat, and the production of a torpid state of mind. Why she seemed to be paralysed on one side I do not know. I think it is probable that these toxins had an effect on one side of her motor area, although not to the degree of causing paralysis. You may ask why is not it the delirium of a toxic condition? I offer this question for your consideration; why is it that the delirium of one disease is not the same as the delirium of another? The idea that delirium was due to raised temperature only cannot be true. Why is it that a patient with acute pneumonia who is delirious is likely to commit suicide? Why is it that when a patient with typhoid fever is delirious he is likely to get out of bed? In the delirium of typhus fever there is no attempt of that kind. I think these various toxins have different effects, and I think you will see that this case is an example of a mental state due to the action on the brain of diphtheria toxins. I could easily mention several other states. But I want to bring before your notice conditions in women often confused with hysteria, and secondly, those conditions which also might be confused with hysteria, but which I believe to be examples of mental disturbance due to a toxin.

*August 24th*, 1908.

# RHEUMATISM.

A Discussion at the Hunterian Society.

**Mr. ROWLAND HUMPHREYS in the Chair.**

Dr. Fredk. Taylor. Dr. Otto Grünbaum.
Dr. F. J. Poynton. Dr. Seymour Taylor.
Dr. Horder. Dr. George Oliver.
Dr. Luff. Dr. Fortescue Fox.
Dr. Fred. J. Smith. Dr. Leonard Guthrie.
Dr. Arthur T. Davies.

(*Concluded from page* 303.)

The President opened the adjourned discussion by referring to what had been said at the previous meeting; he thought there had not yet been any practical application brought forward of the bacteriology of the disease; nor any really authoritative statement as to the length of time during which salicylate of soda should be administered in the disease; nor as to its effect upon endocarditis.

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Information was also needed as to the preventive and active treatment of rheumatic endocarditis, such as possibly might be carried out by a lymphagogue, as horse serum. It would also be interesting to hear remarks bearing upon insurance work; what was the position concerning life assurance of those people who at some time had suffered from acute rheumatism?

Dr. GEORGE OLIVER spoke on the general pathology and balneo-therapy of chronic rheumatism. He said the so-called "rheumatisms" commonly met with in a mineral watering place constitute a somewhat heterogeneous group of painful somatic ailments open to some latitude of interpretation when viewed from the diagnostic standpoint.

Polyarthritis deformans, commonly spoken of as rheumatic gout, is always with us; but this, though usually classed as a rheumatism, is a tropho-neurotic disease depending on infection with degenerative arthritic lesions.

We also now and then see a chronic arthritis (a chronic microbic or infective rheumatism) which is a sort of residuum subacute continuation of an acute infective arthritis. But these cases form merely a small minority of the patients presenting themselves as presumably rheumatic, the 99 per cent. being the subjects of stiffening, painful and deforming affections of the joints, ligaments, tendons, fasciæ, and muscles with localised neuritis and less or more localised arteritis.

The joint affection of this large group of cases is characterised by swellings and nodulosities which are peri-articular, the intra-articular structures, (synovial membrane and cartilages) being as a rule unaffected, at least in the early stages.

*Chronic rheumatism and chronic gout.*—Should these cases be classified as rheumatic or gouty? Fifty years ago this query would have elicited no uncertain answer, for at that time rheumatism was spoken of as either "fibrous" or "synovial," and the term "fibrous rheumatism" would have been assuredly employed.* But to-day the term "gout" is usurping the designation "rheumatism" as hitherto applied to peri-articular and other fibrotic affections of the body and limbs; and the term "rheumatism" is now being more and more restricted so as to cognate a group of diseases (either acute or chronic) of supposed microbic or infective origin with articular, and especially intra-articular lesions. According to this limited connotation of the term we may be said to have very few cases of rheumatism in our health resorts, by far the majority of the painful fibrotic ailments being only quasi-rheumatic or gouty and not strictly speaking rheumatic at all. Surely this conclusion is not admissible. I think that those cases are *gouty* when there is a history (personal or hereditary) of acute gout, or when uratic tophi are present, or when the trouble is apparently self-generated and not dependent on an external exciting cause; while others are *rheumatic* when there is a personal history of an acute infective rheumatism, or when Heberden's or other peri-articular thickenings exist, or when the cause is definitely traceable to cold and dampness, or to overstrain or injury.

*Rheumatism; its seat and nature.*—Over sixty years ago Sir Thomas Watson wrote: "It [rheumatism] is inflammation of a particular tissue—*the fibrous tissue*; and it may therefore manifest itself wherever that tissue is employed in the fabric of the body. . . . Rheumatism therefore is essentially inflammation of the fibrous tissues."* In this statement we have a good illustration of the well-recognised test of truth—its ability to withstand the assaults and disintegration of time. It needs no correction; we have merely to add to it our conceptions of infectivity and auto-intoxication—and we should be reminded that these are not as yet demonstrated facts. Sir William Gowers, in his most instructive lecture on "Lumbago: its Lessons and Analogies," fixes this idea in the term "fibrositis," which he suggests as a designation for all the common forms of rheumatism and unilateral neuritis.† I think this term is useful; it not only clearly indicates the site and nature of the trouble without committing us to any uncertain theory of causation, but it conveniently links together some apparently diverse pathological states into a comprehensive clinical entity, the co-existence of which is a matter of daily observation—such as *local neuritis*, *local fibrosis of arteries*, *gout*, *chronic rheumatism* and *senescence* along with *peri-articular*, or *muscular*, or *tendinous fibrositis*. All newly-coined terms are apt to be resented as clumsy and uncouth; and

* See 'Lectures on the Principles and Practice of Physic,' by Sir Thomas Watson, 1857, vol. ii, p. 741.

* *Op. cit.*, vol. ii, p. 737.

† 'Brit. Med. Journ.,' 1904, vol. i, p. 117.

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that is the only objection which has hitherto been raised to the acceptance of the term "fibrositis." Moreover, this designation may be conveniently qualified by the adjectives "gouty," "rheumatic," "senile," "muscular," etc., and thus it still leaves us free to use the conventional terms "gout" and "rheumatism" in our converse with patients.

*Rheumatic pains, aching, etc.*—There is little doubt that all the pains and aches of rheumatism are essentially due to neuritis; either a local neuritis (fibrositis of a nerve-sheath) or inflammation of the endings of the afferent nerves of the fibrous tissue, which in the normal state is free from discomfort, unless it is unduly pressed or stretched. Sir W. Gowers refers the pains elicited by movement in muscular rheumatism (such as lumbago, etc.) to the compression of the hyperæsthetic muscle-spindles, which are the afferent nerve-endings in the interstitial fibrous tissue of the muscle.* But I think it is possible that pain may also be elicited in a rheumatic limb in another way. We know that when the blood-supply to a muscle is diminished by arteritis obliterans the normal movements may excite intense pain by inducing claudication.† May we not have a local spasm of the arteries which on movement of the affected limb causes severe pain in the same way? Cramps are common in rheumatic subjects and in the early stage of polyarthritis deformans; then there is the frequent occurrence of pain in other local arterial spasms, as in certain cases of Raynaud's disease.

*The immediate cause of fibrositis.*—Apart from such causes of fibrositis as the organisms of infective rheumatism and the autotoxins of rheumatic fibrositis, I shall now discuss some physico-chemical data, which, I think, point to the important *rôle* played by the tissue-lymph circulation in the causation of gouty and rheumatic fibrositis.

*The tissue-lymph circulation.*—The data to which I refer are the outcome of a study of that circulation ‡ which has its seat in the interstitial tissue pervading the body. The physiological and pathological importance of the fibrous or cellular tissue is indicated by the fact that in it lie the meshwork of the capillaries and lymphatics, the nerve terminals, and the cell; showing that it is, as it were, the workshop in which the trophic function of the body is carried on. While on the one hand it receives from the capillaries the pabulum on which the cell subsists, it may, on the other, become a depository of residuary products, which fail to be removed by the lymphatics.

My observations show that under normal conditions the lymph is perfused into the interstitial spaces in a wave-like manner, the flow rising and falling with corresponding variations in the capillary blood-pressure produced by digestion, exercise, etc. In other words, the tissue-lymph does not percolate uniformly on the lines of a static irrigation system, but circulates in variable volume; and this rise and fall in lymph-pressure doubtless serve the important ends of favouring the transfers of supplies to, and of residua from, the environment of the cell.

In chronic rheumatism and gout the data I have observed point to a reduction of this physical interchange, which reduction is probably more pronounced in the area of the fibrositis.

*Deposition of the less soluble residua.*—I have frequently thought that the slackened movement or stagnation of the tissue-lymph interchange may favour the deposition of the less soluble constituents in the lacunæ of the cellular and fibrous tissue. In gout uratic deposits show us that such a process does really go on. It is not improbable that the tissue-fluid, which, however, cannot be isolated for analysis, contains many sparsely soluble constituents, such as the purin bodies, earthy phosphates, oxalates, and carbonates. We know that calcium salts (phosphates, carbonates) are frequently deposited in tissues which are degenerating and in various morbid growths, and that they have been observed as concretions, in appearance like gouty tophi, in the skin and subcutaneous tissue. "These concretions, however, contain no trace of uric acid, being formed entirely of calcium phosphate and carbonate."* Several observers (Drachmann and Stovis, von Noorden and Belgard, Johannessen) have also recorded a low excretion of calcium and phosphorus in arthritis deformans and chronic rheumatism—pointing to the retention of calcium phosphate in the tissues of the body. As yet we have no evidence of the actual deposition of earthy oxalates

* *Op. cit.*

† See Parkes Weber, 'The Lancet,' 1908, vol. i, p. 152.

‡ 'Studies in Blood-pressure,' 2nd edition, 1908.

* 'Metabolism and Practical Medicine,' by Carl von Noorden, translated by Professor Walker Hall, 1907, vol. iii, p. 785.

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in the tissues. Garrod, however, found oxalic acid in the blood of gouty patients, and one exact investigation (that of Mohr and Salomon) shows a low urinary excretion of oxalates in articular gout. I therefore think it is not unlikely that further inquiry will demonstrate the tissue retention of earthy phosphates, oxalates, and carbonates as well as the purin bodies in the various forms of fibrositis.

*Is deposition a cause of fibrositis?*—We have proof of the fact that the precipitation of calcium salts into the lacunæ of the interstitial tissue will cause inflammation; and that has been furnished by several observers (Weber, Jeanne, Morell-Lavallée, Wildbotz, Riehl, von Tannenhain and Levandowsky), who have investigated the cases presenting cutaneous and subcutaneous concretions composed of calcium phosphate and carbonate, or of carbonate only. They found that acute inflammation is excited by the deposits. We also know that now and then the tissues in which urates are deposited become inflamed. May not a diffused precipitate be a cause of fibrositis and of the fibrous proliferations (fibrosis) which it produces? We may suppose that the mere presence of the deposit may only excite a slight degree of local irritation or aching, which is, however, intensified *by moving* the affected part; or it may not produce any discomfort at all, except perhaps a little stiffness, while the part is at rest or is moved in the ordinary way, but *when some active exercise is taken the existence of the deposit may be suddenly revealed by a seizure of intense pain.* Such an event is quite common in rheumatic subjects; and I think the theory of deposition affords a plausible explanation how the tissues are quiescently prepared for the attack of pain and how the sudden invasion is produced. In other cases there may be stiffness or even pain on movement, which, however, ultimately passes off; then the cellular tissue may be dry and becomes freely moistened by lymph exuded by the continuance of the exercise. Perhaps, too, concretionary precipitation may suggest how a draught of cold air causes the forms of fibrositis of muscle and nerve known as torticollis and Bell's palsy; and in the elderly the pains which it incites may reveal where the grit lies.

In fibrositis—whether gouty, or rheumatic, or senescent—the sparse solubility of some of the metabolic products and salts in the blood may make their excretion by the renal epithelium more difficult; and thus their retention and precipitation within the body are favoured. The trend of the most recent work on gout (*e.g.* von Noorden's) is in the same direction.

*Why does humidity favour rheumatism?*—Cold and dampness, especially when combined, are well-recognised causal factors of chronic rheumatism; and observation shows that increased humidity in the atmosphere or in a climate will frequently excite or aggravate rheumatism, even though the air is not cold. Why dampness should have this effect has always been a somewhat vexed question. Just lately a side-light has, however, appeared in the observations of Professor William Thomson, who has shown that less $CO_2$ is exhaled when damp air is breathed than when the air is dry; and that warm dry air has a much more oxidising influence on the blood than damp air.* Bence Jones, I believe, regarded rheumatism as a sub-oxidation disease along with gout and diabetes, and you will remember how often we meet with pains and neuritis in diabetes; and the fact that some of the less soluble residua I have mentioned are sub-oxidation products supports this view. It is, therefore, not improbable that atmospheric humidity favours the development of rheumatism by inhibiting oxidation.

*The balneo-therapy of rheumatism.*—In the few remaining minutes I can only indicate how the main lines of balneo-therapy dovetail with the foregoing conceptions of chronic rheumatism, the aim of the treatment being to remove the effects of the rheumatic process—to ease pain and aching, to reduce deposits and fibrosis, to diminish fibrotic contraction, to remedy atrophy of muscle, and to restore ease and freedom of movement.

*The value of heat and movement.*—The prominent factor in balneo-therapy is temperature, and especially heat, methodised in application and supplemented by other factors, such as massage, electricity and the chemical nature of the bath. Heat is one of our most effective dilators of arteries, arterioles and capillaries, and an active stimulator of the outflow of tissue fluid. *Alone* it overloads and distends for a time the tissue spaces with that fluid; but *in conjunction with motion* (such as passive or active movement of a limb, or massage,

* 'The Lancet,' 1908, vol. i, p. 1093.

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or faradism) it provides the best conditions for the solution and removal of residua—the effusion being enormously increased and the flow from the tissues being greatly accelerated. Thus by the agency of heat and movement the tissue spaces are flushed and rinsed as it were. Hence the value of the following procedures in the treatment of the rheumatic.

(1) Massage douching with hot water (as in the Aix-les-Bains and Vichy douches).

(2) Massage or gentle passive or active movements of the affected limbs during immersion in a hot bath, or at the conclusion of the immersion.

(3) Local hyperthermal air baths (*a la* Tallerman or Dowsing) followed by massage or passive exercise.

(4) Bipolar massage with the high-frequency current.

*Passive bathing.*—When neuritis is a prominent feature of rheumatism quiescent sedative bathing is indicated, such as baths of peat, brine, pine extract, sulphur water, carbonic acid combined with sulphur water or pine extract, or local baths of sulphuretted steam or superheated air. Just lately douching with superheated air has (I am told) been adopted in Germany. It is said that it is not attended by any unpleasant side-effects, and has the advantage of greatly accelerating tissue-fluid exchange, for it deprives the body of an enormous quantity of fluid.

*Internal hydrotherapy.*—Important though the local treatment of rheumatism is, experience shows that general treatment is equally valuable. The benefit derived from a course of baths is often much enhanced by the use of saline aperient waters; and especially of the waters containing the ions, chlorine and sulphur—waters which cleanse the colon, depurate the blood and the tissues by the kidneys, and stimulate the transfers of tissue lymph.

I shall merely add that the bi-weekly irrigation of the colon (*a la* Plombières)* with warm saline sulphur water has, according to my observation, proved useful in those cases of rheumatism in which intestinal auto-intoxication was suspected.

In conclusion, I trust that my remarks, brief and sketchy though they be, may have afforded you some ground for believing that the treatment of chronic rheumatism by hydrotherapy has a fairly justifiable basis in physiology and pathology.

* This method of balneological treatment has been adopted at Harrogate.

Dr. FORTESCUE FOX, speaking of the varieties of chronic rheumatism, said: I propose to briefly describe and distinguish the various chronic affections that are popularly known as rheumatic, and to indicate what treatment by baths and waters is applicable to each of them. Not only the patient, but also the practitioner very probably will, in the early stages of these disorders, be inclined to use this simple, though somewhat vague and much-abused term; but it soon becomes a task of some importance to refer each case to its proper category. and the success of treatment will largely depend on the accuracy of the diagnosis. What is popularly known as rheumatism—and we are now exclusively considering chronic affections—may prove to belong to one of seven or eight categories which are widely different in causation, clinical course and treatment.

(1) In the first place I would refer to the condition Dr. Oliver has already alluded to, which sometimes results from rheumatic fever. This will either take the form of a subacute or chronic arthritic condition with liability to relapse, analagous to the chronic influenzal condition after influenza, or else a purely local thickening and contraction of certain joints. The history of the case amply indicates its nature. As regards spa treatment, I suggest that every convalescent from rheumatic fever should, as a matter of routine, take a course of natural waters and baths. Such a practice would do much to prevent the chronic relapsing arthritis, and also to prevent the more frequent result of rheumatic fever contractions. These local sequelæ are, of course, best treated by warm baths and douches and massage, and form a certain but small proportion of our cases at all health resorts.

(2) Secondly, arthritis deformans (often called rheumatic or rheumatoid) I suggest from a diagnostic point of view that we may know it by its insidious onset in debilitated subjects, and it is so often indefinite. It is thus often only recognised when it has become already far advanced. The type of arthritis deformans varies with the period of life. The progressive generalised arthritis of youth has all the characters of a specific invasion, and the localisation in the joints is only the most remarkable feature of a wasting specific disease.

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This very serious form of generalised arthritis should never be treated by waters or baths, but rather on the same lines as a tuberculosis, with which, indeed, it has many obvious parallels. An open-air life, in a dry sunny climate, perhaps with cool salt baths or douches and the most tonic form of regimen, are the measures which offer the best chance of arresting the complaint. Passing on in life, one comes to the arthritis which is typical of the middle period of life. It is usually a far more amenable condition. Balneological treatment will give us, in many of these cases, the best possible results. It should consist of courses of eliminating waters, repeated for three or four successive years, and on the whole, cold or cool baths with a cool douching and massage. My impression is a very definite one that hot treatment is far too much used in the arthritis of middle life, and that it tends to aggravate the debility and the arthritis depending upon it. And as regards climate, one comes to rely more and more upon cool, dry, bracing airs, the best of which are found on the moorlands. The coincidence of gouty affections in middle life cannot be quite overlooked, and may from time to time modify the line of our treatment. Lastly, we have the arthritis of the aged, generally of far more limited distribution, and often a part of general decay. The circulation and the nutrition should be assisted as far as possible by careful and supporting regimen, and I think that a long-continued sub-thermal course of baths, with or without massage, has a markedly restorative effect in many of these cases. Much depends on the choice of the health resort and the climate, and a sedative air, which does most good in all senile ailments, is especially suitable in cases of senile arthritis. Certain airs, such as that of the north-east of Scotland, are of singularly recuperative value in this condition. It should never be forgotten in the treatment of arthritis deformans, that no bath course is complete without a prolonged after-cure. The neglect of the after-treatment is responsible for the failure of spa treatment in many cases.

(3) In the third place we have as a rare condition, which need not detain us, arthritis ossificans, and if tractable at all, it is probably best dealt with at the health resort by thermal remedies.

(4) In our fourth category, which I suggest is one of very great practical importance to all practitioners, is the gonorrhœal form of arthritis. My experience leads me to think that this is far too often overlooked. The arthritis may be very limited, perhaps confined to one or two tarsal or metatarso-phalangeal joints, although of course the knees are often affected. The wrists usually follow the knee, and ankylosis usually results. But from the point of view of treatment, the urethral condition should be carefully ascertained and remedied, and the resistance of the tissues to invasion fortified by all possible means on the lines of the treatment of ordinary arthritis deformans.

(5) Then we come to the relatively rare but interesting condition of septic arthritis, which often begins acutely in young subjects without cardiac affection. Irregular pyrexias and blotchy eruptions mark the acute stage, and later there is more or less localised arthritis, which may undergo many relapses. But excellent recoveries are often made. Sunny air and the use of brine baths, such as those at Droitwich, seem peculiarly effectual in such cases.

(6) Or we may find that our arthritis is of a tuberculous nature. What is at first considered to be rheumatism may be found to be tuberculosis. But in my experience this is relatively uncommon, while much more commonly what is thought to be tuberculous proves to be gout. The knee is not seldom attacked by gout in young subjects. Tubercular diseases of whatever kind should not be treated by mineral waters or baths.

(7) Seventhly there is gouty arthritis. Wherever there is any possibility of the gouty element in a case of arthritis, waters and baths should be given full trial. Nothing yields so readily to the saline and sulphur and alkaline waters, which promote the elimination of gouty toxins by every route. And warm baths will materially help.

These seven categories are all true arthrites inasmuch as they are severally specific affections of the joints in which synovial cartilages, bones and ligaments are more or less involved. But supposing our case does not belong to any of these; supposing that the joints are not primarily involved although stiffness and swelling and pain on movement may be present; there remains yet another condition upon which I could wish to focus our attention, and that is true rheumatism. Ten years ago I ventured to suggest in my presidential address to the Balneological Society that we should adopt a self-denying ordinance and not use the word "rheumatism" for five years. Now I think that the time has come when we should restore the word and give it an exact connotation. We are suffering at present from terminological inexactitudes in an aggravated form. But I think, sir, that if we could be content to call acute rheumatism *rheumatic fever*, we should find that there is a rheumatism, sometimes acute, sometimes chronic, absolutely distinct from rheumatic fever and from all the categories to which I have ventured to call

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your attention. It is gradually being recognised as a definite entity. Rheumatism in this sense is to be distinguished by its mode of causation and by the tissues which are implicated. It occurs in acute and chronic forms, though the acute of course are liable to pass into the chronic. Chronic rheumatism is a non-febrile disorder, very various in its forms and symptomatology, but pain, stiffness and swellings are always present. It is often described according to the region or tissue which it affects, as lumbago, torticollis, sciatica, brachalgia, muscular rheumatism, periostitis, peri-neuritis. The influence of family history in its causation is marked, and weather, season, climate, soil and locality have also a positive influence. But that which I suggest marks it out as a distinct disease is the ætiological factor. The determining cause is always one and the same. It may be stated without exception that rheumatism is set up by chill. The causation of rheumatism furnishes the best guide to its treatment, which should always include, in some form or another, the application of heat. A homely example illustrates this construction. The man who after sitting in a draughty railway carriage presently suffers from aching in his legs has the essence of rheumatism, and when he returns home and puts his legs in hot water he uses the essential part of the treatment of rheumatism.

Dr. Leonard Guthrie dealt with rheumatism in childhood. He said: I regret I was unable to be present at the last discussion on the subject, therefore I am afraid I may perhaps cover some of the ground which has already been traversed by others. I have no fresh theories to advance, and no original observations to record, and no novel methods of treatment to extol for rheumatism in children. I can only mention points which I think are worth discussion, and I hope to be enlightened myself by the remarks which I shall hear. First as to the nature of rheumatism. My own view is that it is a specific disease and not merely a fortuitous concourse of symptoms as some American writers hold. Its symptom-complex includes pain, tenderness in the muscles and joints, and tendons and nerves, with or without articular effusions. There are erythemata and nodules, recurrent sore throat, pyrexia, malaise, with gastro-intestinal disorder at the time of the acute onset. All these symptoms in children are insignificant, except for their intimate association with cardiac complications, endocarditis, pericarditis and myocarditis. I myself regard chorea as essentially a rheumatic manifestation in children. As to the ætiology of rheumatism in children, I believe that the first factor is hereditary predisposition. It may be, as Dr. Oliver indicated, that there is a rheumatic diathesis, just as we speak of a tuberculous or gouty diathesis. In addition, the presence of some microbic or toxic agent must be assumed, whilst thirdly, there must be an exciting cause in the shape of lowered health and vitality which allows the microbes or toxins or both to have the upper hand. Is there such a thing as a specific rheumococcus? Perhaps this question must be left to bacteriologists to decide. I think their failure to find it in rheumatic cases invalidates the testimony of clinicians who invariably succeed in discovering it. Dr. Andrewes and Dr. Horder may be correct in suggesting that some common saprophytic agent may be at work, the *Streptococcus salivarius* or the *Streptococcus fæcalis*, and that these organisms may possibly become rheumococci in individuals predisposed to rheumatism.

With regard to the minor manifestations of rheumatism in childhood, first of all there is recurrent sore throat. Is there a specific rheumatic sore throat? All will agree that recurrent sore throats are extremely common in rheumatic children and every form of tonsillitis, from simple enlargement to follicular tonsillitis, has been regarded as a rheumatic manifestation. But is there a pathognomonic form of sore throat? My experience and belief is that the most common variety of sore throat in rheumatic children consists in relaxation with congestion of the whole of the fauces, and perhaps some slight catarrhal exudation. What is the bearing of these recurrent sore throats on rheumatism in children? Some regard them as evidence of tonsillar invasion, others as symptoms of rheumatism itself. I think they are merely coincidental, but that they lead to, or are evidence of a condition of, lowered health, and this lowered health may favour the action of the rheumatic poison, whatever it may be. With regard to cutaneous manifestations, I think there is a purely rheumatic erythema multiforme, which I cannot stay now to describe. Erythema nodosum I do not regard as rheumatic. I have hardly ever seen it in association with other stigmata of rheumatism in children. Is there such a thing as a purpura which is rheumatic? I am again in doubt. I think cases which have been recorded as purpura rheumatica have been called so because of the pain and tenderness which are due to effusion of blood about the joints. Epistaxis is also common as an early sign in rheumatic children, and it may be often found in association with articular trouble. Whether it is due to a relaxed and congested condition of the throat I do not know. Rheumatic nodules, I think, are undoubtedly rheumatic. Their varying frequency is of interest. It is a curious fact that although at the hospital the supply of rheumatic cases is fairly constant, many months may pass without one seeing a nodule, and then without any discoverable reason there may be a crop of cases coming shortly after one another. The nodules come quickly and go away quickly. One finds them over all bony pro-

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minences and on the sheaths of tendons; they occur of largest size and most frequently on the superior curved lines of the occiput and on the prominences of the vertebræ. In structure they resemble the vegetations on the cardiac valves, and they appear to begin with thrombosis in small vessels, followed by exudation of lymph cells and blood. Either that exudation becomes absorbed or becomes organised into the familiar fibrous nodules. As to the significance of nodules, one may remember that when attention was first drawn to them some years ago they were regarded as of the gravest possible omen. In my own experience I have never seen them except in association with endocarditis, pericarditis or myocarditis, and I regard them as evidence of an active toxæmic process. But I am not by any means convinced that they are of such grave significance as has been supposed. In some cases I admit that they are speedily followed by a fatal issue, but in many more they subside by themselves and disappear, or they may remain chronic for months, or even for years. As to the age of incidence in rheumatic children, all agree that rheumatism is extremely rare, that is to say, articular rheumatism in children under five. But as Dr. Poynton asks, Why should this be so if rheumatism is due to an infective agent? It is probable the symptoms may be present, but are so transient that they may pass unsuspected. In a series of 52 cases observed by Dr. Poynton under five years of age, 35 were between four and five, most of them nearer five than four, 10 were between three and four years of age, and 4 between two and three, and 3 between one and two, and one who suffered from chorea and endocarditis was only aged ten months. In 96 cases of articular rheumatism under my own observation, I only met with one aged between two and three and two aged from four to five who were sufficiently ill to be admitted to hospital. In the majority of cases seen amongst out-patients under five, the indications and the history were extremely vague. Sometimes by the time one sees the child they have disappered. With regard to the diagnosis of rheumatism in children under three—scurvy, congenital syphilis, poliomyelitis, osteomyelitis have to be excluded, also tuberculous arthritis and septicæmia. In children under three, cardiac signs are more frequently due to congenital malformations than to endocarditis, whilst choreiform movements under three years of age are far more suggestive of gross cerebral disease than of true chorea.

With regard to treatment of rheumatism, were it not for the imminent practical danger of myocarditis, endocarditis, and pericarditis, articular rheumatism in childhood would be a very trivial affection. Children under twelve rarely pass through six weeks of intolerable agony, as many adults do. In children articular rheumatism usually subsides in forty-eight hours after rest in bed and warmth to the joint and the administration of sodium salicylate. The temperature falls and the effusion subsides. In the out-patient department the pain has often disappeared before the patient is seen. Any evidence of it having been recently present and especially any indication, however slight, of cardiac implication should be treated at once by rest in bed for at least three weeks. I need hardly emphasise the danger of allowing these children to run about and of forcing them to go to school. Though I speak of putting these patients to rest in bed, I must say that in my experience rest in bed does not always avert the danger of endocarditis. And yet one must admit in common sense and reason that a child who is allowed to go about in these conditions is far more likely to be rendered incurable than is one who is kept under proper control. I have no doubt salicylates relieve pain and reduce the temperature in a few doses in young children. But are they specific cures, including the much-vaunted aspirin? I do not believe that they prevent cardiac complications. I have seen these latter conditions arise in children who have been fully under the influence of salicylates. If salicylates are specific I should be one of the first to agree with those who administer enormous and even heroic doses, but I have never been able to discover from those who give these heroic doses that the cardiac troubles are prevented. And during the administration of salicylates it is not uncommon for the nodules to appear. It is said that if sodium bicarbonate is given in combination with salicylate of soda any ill results may be prevented. I am not at all certain that that is so. The symptoms of salicylate poisoning are those of fatty acid intoxication: thirst, air-hunger, vomiting, acetonuria, the presence of acetone in the breath, and coma, which coma may even end in death. In such cases of acid intoxication following chloroform anæsthesia as I have seen the symptoms are not in the least influenced by sodium bicarbonate, and therefore I doubt whether we are justified in saying we can prevent ill results of large doses of salicylate of sodium by giving sodium bicarbonate at the same time.

With regard to carditis, I cannot go fully into the matter, although it is of the greatest importance in connection with treatment, and the treatment of rheumatism of course means the treatment of endocarditis and pericarditis in children. If one considers the pathological appearances after death in a case of carditis in rheumatism one cannot feel surprised that treatment was in vain. If myocarditis is present, the commoner course of giving digitalis to all cases I think is absolutely wrong. The action of digitalis on the heart when myocarditis is present is useless, and the test of the good or ill of digitalis is afforded by the pulse. If that remains quick,

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digitalis is doing no good. If the pulse becomes hard and small and regular it is doing harm; and there is danger of it causing acute distension of the right heart, owing to increased resistance from contraction of the arterioles which it produces. But one admits that acute distension of the right heart, which is a common and serious complication in children, may occur from mitral regurgitation and backward pressure when no digitalis has been given, and in such cases digitalis combined with squill and mercury is of great value. It should be preceded by bleeding or leeching or dry cupping as the case may be. I have used theocin sodium acetate with good results in cardiac dropsy. But it frequently has the effect of producing serious vomiting, and it is not superior to the old-fashioned Baley's pill or powder. In pericarditis, blisters may seem to relieve pain, but I am not certain that the relief is not due to effusion of fluid which prevents the inflamed surfaces of the pericardium rubbing together. Hot applications sometimes relieve it, and Dr. Lees recommends the use of the ice-bag. On one occasion I used it, and I thought it was of service. But when there is much pain and restlessness morphia is the only drug which we can give with good effect. As to chorea, is it a rheumatic manifestation? The reasons for thinking so are, first of all the frequency of personal or past history of rheumatism, and then a family history of it and cardiac complications which we recognise as rheumatic. In 114 of my cases a family history was marked in 47·3 per cent., absent in 42·1 per cent., doubtful in 9 per cent.; there was a personal history marked in 63 per cent., absent in 32 per cent., doubtful in 4 per cent. Cardiac complications were marked in 44 per cent., absent in 42 per cent., doubtful in 4 per cent. But in chorea not only are the factors of rheumatism which I have mentioned of paramount importance, but still more important is the neurotic element, that is to say emotional instability, which affects all children who suffer from chorea. The absence of a neurotic temperament accounts for rheumatism without chorea. We know that children may go through courses of acute rheumatism without suffering from chorea, but those are not neurotic children. The absence of rheumatic symptoms in chorea does not prove that chorea is not rheumatism, for in many cases choreic children may develop rheumatic symptoms afterwards. Dr. Batten followed up a large number of chorea cases treated at Great Ormond Street Children's Hospital, and showed that 20–30 per cent. of them, though they had no symptom of rheumatism while the treatment at hospital was being carried on, developed these symptoms within six years afterwards. As to "fright cases," it should be remembered that fright is only another name for shock, which may be emotional or physical. Any traumatism, alarm, or shock occasioned by getting wet through will so lower the vitality of the person that it will give the rheumatic poison the upper hand. I think chorea is a manifestation of rheumatism in the higher cerebral centres in an emotional individual. The symptoms point to psychical disorder rather than to irritation of the motor areas alone. The movements are not spasms or convulsions, and therefore are not movements which may be produced by the irritation of the motor area of the cortex alone. In acute chorea the movements suggest uncontrollable impulses to perform violent contradictory actions at one and the same time. The facial movements are exaggerated, expressions of all the emotions quickly succeeding and clashing with each other. These expressions of the emotions are not produced by experience of the emotions themselves. Some define chorea as a loss of inhibition on higher over lower motor realms. Doubtless there is such loss of inhibition, but it is combined with an uncontrollable impulse to perform choreic actions, and I think this has some bearing on the treatment of the complaint.

As to the stages from the point of view of treatment, we have first the acute uncontrollable stage. Its duration seldom exceeds three weeks; it is generally only a week to ten days. This may be followed by the paretic stage, in which the movements practically cease, or are only represented by slight jerks or twists. Extreme prostration and very often mutism are present. The child develops a fatuous mental state, there may be delirium, night terrors, crying fits, mania is not uncommon, and the period of this stage exceeds that of the uncontrollable acute stage. We have another stage or termination, in which the movements still continue, but they are capable of control by the will. Sometimes the movements continue only when the child is under observation, sometimes they only occur when the child believes itself unwatched. With this there is also marked ataxy on voluntary action The duration of this residual or habitual stage of chorea may be unlimited unless it is properly treated. In the acute stage, rest, quiet, protection from injury, and isolation are necessary in the treatment. There should be plenty of good food, and it may be necessary to carry out nasal feeding. When the movements were very violent I have thought that splints were of considerable service. The paretic stage requires abundant stimulation and good food, in fact the paretic stage should be treated in the same manner as the stage succeeding any exhausting illness. Massage is invaluable. I have seen electricity do harm by frightening the child. The residual stage is ascertained by testing the power of voluntary restraint which the child possesses, and the treatment is to encourage the child to exercise that power. One uses the Kindergarten apparatus and other similar means to improve and encourage the precision of movements.

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With regard to the employment of drugs in chorea, I think but few people believe in arsenic now. Twenty years ago I had the opportunity of watching the effect of the exhibition of arsenic in every possible fashion, both small and large doses, and on the intensive method. I have never seen it do good in large doses, but frequently I have seen it do great harm, producing not only gastro-intestinal irritation and vomiting, but also the far more serious condition of arsenical peripheral neuritis subsequently. I admit that the movements of chorea in the acute stage will often cease under its use, but they will also cease spontaneously without any form of treatment. I admit some children tolerate arsenic, but I suppose that in such cases the drug is not absorbed. With regard to sedatives and hypnotics, I prefer chloral and bromide to any. I have heard people say that chloral is apt to produce mania in chorea, but as mania occurs in chorea without the use of it I am not convinced that the drug has much to do with the mania. Bromides alone I do not think are of much service. Antipyrin was much recommended ten or fifteen years ago, and I have tried it, but without any considerable success. With regard to salicylates and aspirin, salicylates, I think, are useful whenever pain is present, and in cases of chorea it often happens that pain is present. I do not mean in the way of arthritis; choreic children, fortunately for them, do not have chorea and articular rheumatism at the same time, but they complain frequently of pains in various parts, and in such cases salicylates and aspirin, which is acetyl-salicylic acid, are of equal service I think. With regard to large doses in chorea, I am absolutely against them. I have been struck by what I have been told by those who have used large doses: they seem more impressed by the fact that some children can take them with impunity than able to prove that the children are any the better for doing so. Aspirin has been highly spoken of by Dr. Bligh-Wall recently, Dr. Burnet and others, but what strikes me about the reports of these cases treated by aspirin is that it seems to be a matter of remark and wonder that the great majority of them are cured in less than three months. But to my mind the case which is not cured of chorea, or does not cease from having it in that time, is no testimonial to any form of treatment which may have been adopted. My own experience of chorea at Paddington Green Children's Hospital is that it does not exceed four to five weeks in duration, although that is much less than the time given by other observers. The conclusions offered are that rheumatism is a specific infective disease. Secondly, that the factors concerned in its production are, first, inherited predisposition, secondly, the presence of an undetermined microbic or toxic agent, which may possibly be innocuous in non-rheumatic subjects. Thirdly, a condition of lowered vitality in the subject, which may be introduced by sudden shock of any kind or prolonged exposure to cold or damp. Chorea is a rheumatic manifestation, which occurs only in those who are emotional or neurotic. It is a psychical disorder. Its causes may be emotional disturbance, in addition to the factors concerned in the production of rheumatism itself. There is an acute stage of chorea, characterised by an uncontrollable impulse to perform apparent contradictory movements, with a duration of one to two weeks, a paretic stage accompanied by profound physical prostration and mental disturbance, with cessation of choreic movements, or one in which the movements continue, but can be restrained by active will. Drugs may palliate, but I do not think they cure rheumatism, or chorea either. The treatment of acute chorea which is most successful is rest, quiet, sedatives, and salicylates when pains are present. The paretic stage should be treated on ordinary principles. The residual stage needs treatment by moral persuasion, drills and exercises. The practice of poisoning choreic patients by arsenic and salicylates is deprecated. The general prostration induced thereby may appear to shorten the acute stage of chorea. Bacteriology seems to afford the only ray of hope in the matter of treatment, and I think we must put our trust chiefly in the use of appropriate vaccines, which may hereafter be invented.

Dr. DAVIES said: Dr. Frederick Taylor asked a question with regard to the bearing of rheumatism on life assurance practice, a matter which I am interested in. I hope he will tell us what he does himself in these cases. It is very difficult to assess the life of a man who comes before you with a history of acute rheumatism. I think you may divide these people into two classes: one in which there is a family history of rheumatism, and one in which there is not such a history. The man who has a history of rheumatism in his family I should look upon with greater suspicion than one who has not, and I adopt the principle, if possible, of putting him on what I call the limited payment principle, with extra premium. I do not think I should take that off, even after several years. But in a healthy man who has a healthy heart, but who has had rheumatic fever, I would rate him up, if only for a few years. After ten years one might put him on the usual rate. I have been struck by this matter on looking over the claims in my office; I have noticed how very few claims seem to emerge as due to heart disease following acute rheumatism, even where there was anything wrong with the heart at the time of examination for the policy. And I think one is apt to be unconsciously biassed in one's hospital work otherwise by the fact that people in the lower classes are so much more liable to get recurrent attacks of rheumatic fever than are

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those in a better social position. A man in a good position is more entitled to be let off lightly in the matter of premium if he has gone some years after rheumatism without damage to his heart, than is a man who has also had rheumatic fever but who belongs to the labouring classes. With regard to treatment, which is of course the apex of our discussion, I wish to refer to acute rheumatism, or rheumatic fever, and not to what Dr. Oliver and Dr. Fortescue Fox referred to. If we ask ourselves whether we have advanced a bit further in this matter than in the days when salicylates were first introduced, or if the old six weeks' cure of rheumatic fever is as true now as it was then, we can answer that we believe it is. We admit salicylates reduce the fever and the pain, but they have not reduced the cardinal lesions, such as endocarditis. The condition in regard to them seems to me to be the same as it was before. Sir Douglas Powell, in his Lumleian Lectures, said he thought that the salicylates had not been fairly treated in this respect. He says: "The infrequency with which aortic regurgitation and mitral stenosis are met with in primary cases of acute rheumatic fever cannot fail to have impressed the clinical observer, but the lesson to be received from this fact has not been duly learnt." He says that Dr. Sampson points out that in rheumatism treated in twenty-four hours by rigid rest in only one case was there aortic mitral disease, whereas in 19 cases out of 127 not so rigidly treated in this respect aortic regurgitation was present. He says that even Dr. Sampson did not point the lesson, for he only insisted on the importance of prolonged rest for a few weeks after the acute attack. But Sir Douglas Powell says that they should be rested longer than the persistence of the heart mischief. But it is impossible to do this. It may be that patients are not kept sufficiently at rest to get the result we wish for on their heart. I was struck by Dr. Guthrie's remark that rest is of little or no effect in the endocarditis of children. [Dr. Guthrie: I said it did not prevent it.] I would remind the meeting of the treatment by blisters, which was introduced by my father, Dr. Humbert Davies, and he maintained that blisters should be applied over and around the joints, and that they had a tendency to prevent endocarditis. Dr. Cave, of Liverpool, has modified the treatment by putting small blisters on different areas over the heart, so as to prevent endocarditis, and he seems to think it has that effect.

The PRESIDENT: Dr. Horder wrote to me a few days ago a letter in which he pointed out that Dr. Poynton had had an opportunity to reply, and he, Dr. Horder, claimed the same privilege. Dr. Horder said he would have explained his use of the term "malignant endocarditis," so severely criticised by Dr. F. J. Smith, who was supported in his objection by Dr. Poynton. He says, "I am convinced we are dealing with a second chapter when the endocarditis takes on the ulcerative or fungating form; that the micro-organism is isolated from the patient at this second stage, and that it has no necessary causal connection with acute rheumatism." I do not know whether the production of leucocytosis has been tried in acute rheumatism. But in rheumatic fever we have a natural leucocytosis, and it has occurred to me that such a drug as nucleic acid might be administered, which increases the leucocytosis and so increases the resistance of the organism to the action of the rheumatic causative agent. I think it is essential that we should be able to have some definite statement this evening as to the action of salicylate of soda, and whether we are to rely upon it as an antidote to the disease, or whether we are only to look upon it as a reliever of pain. Personally, I tell my patients that it relieves the pain but does not cure the disease. I have looked upon it as simply an agent in relieving the pain of the disease, and the patient, of course, has to stop in bed. The trouble with patients suffering from endocarditis is that they will not remain in bed after the pain has been relieved.

Dr. F. TAYLOR, in replying, said: I daresay you will not expect me to answer in detail all those who have spoken. Those who heard my address will remember that I was trying to confine my remarks to conditions which could be called rheumatism in the most restricted sense of that word; and therefore I did not desire to say anything about any of the chronic articular affections whose connection with acute rheumatism was most doubtful. Nor, indeed, would it have been possible for me, in the time which was allotted to me, to have gone at all fully into any one of those subjects if I discussed acute rheumatism itself in detail. So far as I am asked to answer remarks which have been made in reference to my paper, I may say that many speakers have not dealt with it at all. I was followed by Dr. Poynton and Dr. Horder, and I hoped to hear from them some settlement of the vexed question as to whether rheumatism is, or is not, a bacterial disease, and whether the bacterium has been discovered and identified. It will be remembered that there was a difference of opinion between them as to the origin or cause of rheumatism, and that Dr. Poynton, in his reply, declined to accept the explanation of his results offered by Dr. Horder, and maintained his position that there was a definite *Diplococcus rheumaticus*. Dr. F. J. Smith was kind enough to express his approval of much that I said in my paper. I remember his attack on the use by so many writers of the term "malignant endocarditis," and though that was only a side issue in regard to anything I said in my paper, I thought I might now make a few remarks about it.

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I admit that the term "malignant endocarditis" has to be defended. I have in my hand a paper which I published seventeen years ago,* entitled, "Malignant Endocarditis"; and I devoted in that paper a page and a half to the subject of nomenclature. I will not inflict the reading of it upon you now, though much of it remains true at the present time. The point, it seems to me, is that we want a name to represent the fact that the endocarditis is doing something beyond its immediate locality of the valves. What we should name is the general condition of disease of the whole body, with endocarditis as supplying the qualifying epithet The old term which Sir Samuel Wilks employed, "arterial pyæmia," would be a better name, or "arterial septicæmia," or "cardiogenous septicæmia," or "endocarditic septicæmia." For it is, in fact, a septicæmic condition which has its origin in infective disease of the valves of the heart. But I think the term "malignant endocarditis" is justified as expressing an endocarditis which is malignant in its operation by its widespread effects, in the same way as we speak of malignancy in connection with the widespread effects of cancer and other allied diseases. The terms "infective" and "infecting endocarditis" would suggest possibilities, but the terms "fungating" and "ulcerative endocarditis" only express the condition of the valve itself.

Dr. Luff gave us an interesting account of his views with regard to very much the same branch of the subject as Dr. Oliver dealt with to-day, that is, what has been called by the very ugly and unsatisfying name of "fibrositis." I have not ventured to deal with these things, but only with acute rheumatism itself, and therefore I do not feel bound to discuss this evening the actual nature of the lesions which have been called "fibrositis." I submit we do know very little of their origin; and the chills, depression, or autotoxins to which they are attributed probably have little connection with true rheumatism. We have to be satisfied with recognising that under many circumstances we are subject to local inflammations of fibrous tissue—sometimes muscle, sometimes tendon, sometimes ligaments, sometimes synovial tissues, but I think we ought to try and keep these in our minds as distinct from acute rheumatism. I was pleased to hear Dr. Oliver's ingenious remarks with regard to the production of this fibrositis. He would allow, I take it, that he has not got the completest possible evidence that everything that he suggested is at present the fact. But there is a possibility that some of those complaints may have arisen in the way he suggested, namely, the deposition of salts under certain circumstances in muscular and fibrous tissues. I should like to ask him what positive evidence he has of that occurring, whether he has seen the salts in the tissues, other than urate of sodium, of course. Dr. Fox has dealt with the subject very largely from the clinical side, and he has not told us about the treatment of rheumatism so much as about the treatment of arthritis in general. He included many forms of arthritis which are not rheumatic; for instance, gonorrhœal rheumatism, which is out of the scope of our discussion. We know that in gonorrhœal arthritis there is a specific organism present; and septic tubercular and gouty forms of arthritis are not rheumatic. There can be no doubt about the value of hydrotherapeutic treatment in the cure of some cases of rheumatism, but one surely lives in hopes at the present day of something a little more specific than the treatment which we find so useful in many cases at the various spas and bath resorts. One would like to prevent the necessity of such treatment by a more specific clinical treatment than we possess at present. With regard to Dr. Guthrie's remarks, I began to think he might have written my paper instead of me. I am generally in sympathy with his treatment of the subject. He has shown what I consider very proper scepticism with regard to the relation of various complaints to rheumatism and the influence of some kinds of treatment. We must admit there is a very great deal indeed to be learnt, and that the subject is not so hopeful as we often try to persuade ourselves. In my paper I commented on the salicylate treatment and asked the question what effect did it have in the prevention of endocarditis? And my general impression, though I was not speaking upon very extensive statistics, was that there was no evidence that cases of heart disease had diminished in the last thirty years in consequence of the abandoned use of salicylates. It would be impossible for me at this hour of the evening to deal with the many details which Dr. Guthrie gave us in regard to rheumatism in children; I should only like to express some doubt with regard to the question of diathesis, whether the rheumatic diathesis has much to do with the outbreak of the disease. Unless there are statistics which have been very carefully collected and analysed, we should be very scrupulous about accepting statements with regard to rheumatism in parents and grandparents, such as we frequently get in collections of figures on this subject. Also, I am not prepared to regard chorea as essentially psychic in character, which I think was the suggestion Dr. Guthrie made. It think it is a cerebral disease, but that the first effect is on the motor centres, and in certain cases it extends to the psychic areas of the brain. I do not doubt that the patients have definite mental characteristics, and in many cases get maniacal, and even in the slighter cases there is deficient mental capacity. But I cannot regard the motor defects as being the results of psychic failure. I think they are simultaneously produced.

* 'Guy's Hospital Reports,' 1891, vol. xlviii, p. 171.

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# THE USE OF VACCINES.

By W. BRUCE CLARKE, F.R.C.S.,
Surgeon to St. Bartholomew's Hospital.

GENTLEMEN,—As you know, I have been employing vaccines in the treatment of many cases both of acute and chronic disease, especially during the last few months. The results have been good, in some instances very striking, and I propose to discuss some of these cases in my clinical lecture to-day.

The first case to which I propose to refer is that of a man, æt. 24 years, a furrier by trade. The history of his illness is a curious one, and its termination equally striking.

He was in perfect health up to July 10th of the present year. That evening he went to bed as usual, but was seized during the night with a severe rigor, followed by frequency of micturition. The following day he managed to get up and go to work, though he felt very ill. Micturition was very frequent throughout the day; he was passing water every half-hour, suffered considerable pain when he made water, and on one occasion passed some dark fluid, which he believed to be blood.

The frequency, pain and malaise persisted until his admission into Sitwell ward about a week later.

When the first note was taken of his case on the day after admission his frequency and pain had not altered since his admission. His prostate was tender to touch, but not markedly so, and he had in addition some pain, but not of a severe character, over both his kidneys.

The urine contained no trace of pus, albumen or blood. The diagnosis was obviously doubtful; but as he had passed some fluid which might have been blood when he was first taken ill, a cystoscope was passed. The bladder generally was healthy in appearance, but there was some congestion near the urethral orifice.

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His temperature rose to 102° F. and remained several degrees above normal for several days, and the pain over his kidneys increased in severity. He had various drugs administered but without any beneficial effect.

When I saw him about a week after his admission his condition was obviously becoming more serious; he looked very ill and was in considerable pain. His urine had been examined for micro-organisms and was found to contain abundant *Bacillus coli*.

I directed that some *B. coli* antitoxin should be given without delay as I felt there was no time to be lost, and gave orders for a vaccine to be prepared from his urine, and injected as soon as it was ready for administration. The preparation of a vaccine demands, as you know, four days at least.

Twenty-four hours after the serum had been administered the temperature began to fall, but it did not come down to normal, and he still remained in a somewhat serious condition. I wish to draw your attention especially to this fact, because it contrasts so markedly with what occurred when the vaccine was injected, which you will remember was prepared from his own urine.

*Within a few hours his temperature fell to below normal and it has not risen since.* When I went round Sitwell ward just a week after the patient had been in the serious condition which I have just described to you, he said he felt perfectly well. He looked in good health, and was anxious to be allowed to get up. A day or two later he got up, and a few days afterwards went home. I was anxious to keep him in the hospital a short time longer as the urine, though in appearance perfectly normal, still contained some *B. coli* in small quantity, but he insisted on returning home as he was anxious to go to work.

A week later he presented himself for readmission as his symptoms had begun to return. He felt weak and ill, his urine was again cloudy, and he was troubled with a frequent desire to pass it, which amounted almost to incontinence.

He was at once put to bed and treated by the frequent administration of diluents, together with citrate of potash and urotropine in 10 gr. doses every six hours. No further vaccine was administered. Under this treatment he has steadily improved, and during the last three days no trace of an organism has been discovered in his urine. To-morrow he leaves us, this time I hope permanently cured.

I have dwelt somewhat at length upon this case, because it illustrates very fairly the mode of onset of an acute attack of *B. coli* infection. I will refer to the chronic variety of this disease presently.

These very acute cases are rare, but in years gone by I have seen a certain number of them, and their death-roll is a very heavy one. The patient may be seized with a rigor without any warning, and within a few hours be attacked with all the symptoms of a very acute cystitis, and die in three or four days in spite of the most energetic treatment, in some instances with almost complete suppression of urine. Twice I have seen this condition supervene upon an ordinary attack of gonorrhœa, and it was followed in both instances by death in less than four days. It is only within the last few years that I have realised that these cases were the result of *B. coli* infection, and perhaps I am wrong in supposing that no other infective organism can produce similar symptoms. What I do know is that whenever I have had the urine of these cases bacteriologically examined the pathologist has invariable reported the presence of *B. coli*, and has generally further remarked that it is not quite the ordinary form, as it does not answer to all the usual tests. I am inclined to suspect that we are dealing with a special and highly malignant variety, the life-history of which I have no doubt we shall become better acquainted with some day.

As I have alluded to the subject of energetic treatment, quite apart from vaccines, you will naturally demand what I mean exactly by these words. Under this heading I include abundant diluents accompanied by sharp purgation and large doses of quinine. Dry cupping over the loins should be resorted to and repeated. If the attack does not show signs of abating, the kidneys should be explored, and an incision made into both of them, so as to arrest suppuration which is almost certain to occur if the patient survives for a long enough period. It is because of the very serious nature of these acute cases that I have your special attention to them and to the striking effect which has been produced by a vaccine in this one.

How did the infection reach the urinary tract—for it appears to have been confined to it alone? I

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cannot give you any certain answer. It looks as though a sudden dose of virulent *B. coli* infection had found its way by means possibly of a temporary injury, that had for the moment joined the colon to some part of the urinary tract. I have known cystitis to be developed during the course of a case of sloughing piles, but never one that gave rise to dangerous symptoms. Suppose, for example, a bone in its passage through the bowel had contrived to temporarily wound the bladder, I can conceive that it might give rise to such an attack. But I will not pursue this side of the question; we are wandering into the region of mere conjecture.

But if it is rare to meet with cases of this acute type, it is comparatively common to come across those of a milder form, and to this class of case we will now direct our attention. For want of a better term I will call these cases chronic. In one sense it is an unfortunate term, as we must include under this heading cases which vary vastly in their duration and severity, but like all chronic cases they are far more difficult to cure. Vaccines will do wonders for them if persevered with, but they will probably last for months, and certainly will not yield to a single dose of vaccine.

Before I allude further to this group of cases I should like to caution you against taking them too seriously, patients may have *B. coli* in their urinary tracts and live a happy and fairly comfortable life for many years. Remember I am not suggesting you should ignore these cases or dismiss them as unduly trivial, but only cautioning you against giving too serious a prognosis.

Only the other day I saw a lady on whom I did a colotomy between eleven and twelve years ago, because she was passing wind by the urethra. She had a fistulous tract between some portion of her intestinal tract and the interior of the bladder. I could see the opening in the latter quite easily by the aid of a cystoscope, and note the little bubbles of gas issuing from it. These cases are always the cause of considerable discomfort, and oftentimes produce acute attacks of cystitis. I opened the abdomen of this patient and found her pelvic contents densely matted together. She was fat and subject to attacks of bronchitis; after I had explored her pelvis as much as I dared, I found no trace of the intestinal opening as I hoped I might have done, so I decided to perform a colotomy.

Before the operation she often passed slight quantities of fæcal matter with her urine, and though as a rule she is absolutely free from any urinary trouble at the present time, yet the urine is never free from colon bacilli. She is in much better health than she was before my operation, and has learned to regulate her bowels so well that her colotomy wound gives her but little trouble.

Many a man who has been in the habit of using a catheter once or twice daily constantly has colon bacilli in his urine, and seems to suffer but little or no inconvenience from the condition.

A chronic case of colon cystitis has usually had an insidious origin. In all probability when one is first consulted for this condition by a patient, he states that he has pain somewhere about the neck of his bladder for some time, or he has suffered from some irritability about his bladder with frequent calls to pass water, and probably he has been in the habit of passing a catheter. Suddenly this condition, which has almost passed unnoticed and is only recalled perhaps when one carefully cross-examines him, becomes aggravated, and he realises it is time to consult a doctor. One naturally makes an examination of the bladder region, as it is of the bladder that he complains. The lower portion of the abdomen may be tender to palpation, possibly deficient in movement, or intolerant of pressure. If one makes a rectal examination the prostate is almost certain to be unduly tender; if the patient's symptoms are severe the tenderness will be extreme. His urine will almost certainly be acid and may be quite clear. If it is cloudy there may be flakes of lymph floating about in it. There is rarely much in these chronic cases so far as the naked-eye appearance is concerned, and it may be difficult to find bacteria until the urine is centrifuged. As a rule this is comparatively easy when one of these more or less acute attacks has supervened upon a more chronic condition.

A few polymorphonuclear leucocytes will probably be present, and there is most probably a slight cloudiness on boiling. The colon bacillus is usually not a typical representative of the species. It does not answer to all the typical tests. I want you particularly to note this fact, because it has an important bearing on the treatment of the case.

You will remember that I have already drawn your attention to the fact that in the first case, an acute one, which I described to you, the result of

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the vaccine treatment was far more marked and much more rapidly manifested than where the serum was administered. The difference is this, the serum is prepared from an immunised animal, the vaccine from the patient's own secretions. In the case of a serum there must always be some doubt as to whether it will be all-potent against the offending bacillus; in the case of a vaccine all doubt is set at rest, and here lies its great superiority, especially in these urinary infections.

The next case which I am going to refer to affords an excellent illustration of this point, though it turned out not to be a *B. coli* infection.

The patient, a man, æt. 27 years, came into the hospital in consequence of some frequency of micturition, coupled with pain over the pubes. His temperature was slightly raised but he was not seriously ill. His condition answered to the general symptoms which I have just described to you. His urine was acid, almost clear, 1018, and contained but a trace of albumen. There was no stricture and no history of an attack of gonorrhœa to be obtained, so that the cause of his illness was far from clear. His urine was found to contain bacteria, but they did not answer to the ordinary tests of *B. coli*. Without waiting for an exact diagnosis I directed a vaccine to be prepared, as it appeared the case was of recent origin, and I had every reason therefore to hope, as in the first case, for a speedy cure. The vaccine was prepared and administered to the patient with the very best results; in ten days he was perfectly well. The vaccine had almost done its work before we obtained a report from the pathological department, which informed us that it was Gaertner's bacillus which had produced the attack of cystitis. This is one of the first cases of the kind which I have come across.

My house-surgeon, Mr. Lang, who is, as you know, a great enthusiast on the subject of vaccines, has been employing them in a variety of the cases of minor surgery, which do not as a rule remain long in our wards, and with the happiest results. I have been much struck by the rapidity with which the healing process sets in.

In one case, a male, æt. 39 years, a carbuncle on the neck supervened upon a case of acute *Staphylococcus pyogenes* cellulitis of the hand. The carbuncle was a couple of inches or more across. A vaccine was prepared from the hand and injected. A single dose produced a marked effect on the carbuncle, which was only superficially incised. Forty-eight hours after the incision it began to diminish in size and assume a more healthy appearance. On the fourth day the slough began to come away, and had completely separated on the sixth, whilst a week later it had practically healed.

Another of our patients happened to be suffering from ciliary blepharitis, which, if you have dressed in the eye-ward, you will know is often not very amenable to treatment. The woman had had her eyes bad for some years, and derived so little benefit from the various eye hospitals at which she had attended that she had made up her mind not to trouble any further about the matter. She was vaccinated on two occasions, and her eyelids resumed their natural condition. Three months later she got another attack and so impressed had she been with the results of Mr. Lang's treatment that she promptly returned and demanded to be vaccinated (or injected, as she called it) again. Her request was acceded to and the result was successful.

Let us return, however, to the further consideration of these *Bacillus coli* cases, for I want to refer to another peculiarity which they present. They do not always limit themselves to the urinary tract. They spread up to the vesiculæ terminales, which are often tender and inflamed during the course of an attack, and if this is the case you should be on the lookout for further complications involving the testicle, epididymis, and cord.

It is rare, so far as my experience goes, for true suppuration to occur, but fluctuation may be felt and incision possibly made in the testicle under the impression that pus will be found. The fluid which is evacuated under these conditions is usually nothing but blood-stained serum. I believe that this is characteristic of a coli infection of the testicle.

But the trouble may not end here: the veins of the leg may be swollen, so that the patient appears to be the subject of an ordinary attack of phlebitis. Whether suppuration ever occurs in these phlebitis cases I cannot say. I have never known it to occur, but I should certainly not be surprised if I came across such a case.

So far I have alluded only to this affection as it occurs in the male sex, but it is certainly not con-

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fined to them. Indeed, cystitis of colon bacillus origin is far more frequent in women than in men, but as a rule it is in them a comparatively mild disease.

It is no infrequent event if you examine the urine of women apparently in perfect health to find colon bacilli present, and yet no frequency of micturition or any other clinical symptom marks their presence. This is perhaps not peculiar if we reflect for a moment on the anatomical differences of the two sexes so far as this portion of the body is concerned. When clinical evidence of the disease is forthcoming in the female sex, it almost always takes the characters of pelvic peritonitis. It betrays itself by some tenderness, more or less pronounced, of the pelvic contents. The uterus and ovaries may be tender to the touch, and there is often painful menstruation as well. Indeed, I think I may go so far as to say that pelvic peritonitis in a woman is rarely present without some degree of cystitis.

Before I pass on to consider the general indications for treatment in these cases, I should like to relate to you the history of a case which has been under my care during the last few months. The patient, a man, came to me some nine months ago with a well-marked colon cystitis and an enlarged prostate. It is needless to tell you that he had been treated by catheters before I saw him. When he first came under my care he was passing water every half-hour during the day, and at frequent intervals during the night as well. I examined his bladder under an anæsthetic and measured his residual urine, which varied somewhat, but rarely exceeded 2 oz. in amount. His prostate was not apparently much enlarged. I decided to treat him by washing out his bladder, diluents and the administration of urinary antiseptics, and to keep him, if not continuously in bed, at least in the house and in a warm room. He improved somewhat, but his temperature was raised frequently at night. He did not make the progress, I must own, which I had hoped he would. I was loth to remove his prostate, because I deemed it a somewhat hazardous proceeding in a man of his age and condition, and moreover, the prostate, as I have already told you, was not much enlarged, and the residual urine small in amount. Vaccines seemed powerless to effect a cure, or even to afford him much alleviation.

As, however, his symptoms did not disappear, and as he was clamorous that something should be done to relieve him of his trouble, I at length decided to perform a prostatectomy. I need not trouble you with the details of the operation; it presented no special difficulties, and his subsequent recovery was uneventful, except that he recovered with greater rapidity than I had expected, and seemed to begin to pick up as soon as the prostate was removed. But the curious part of the case, to my mind, has yet to be related. Three weeks after his bladder wound was healed the urine which he passed was perfectly healthy, and showed no trace whatever of colon bacilli. The reason I am specially drawing your attention to this case is because if this case is to be regarded as a type, it would seem as though the prostate was the main seat of infection in these cases, and as though its removal might be regarded in many cases as an important adjunct to our means of treatment in many of them. Further, it enables us to understand another fact to which I have already alluded, viz. that colon bacillus infection is a far less serious affair in the female sex than it is in the male.

And now let me revert more in detail to the general lines of treatment which you should pursue in these cases. Though I have devoted this lecture mainly to colon infections, remember the principles are the same whatever the variety of infection with which we are dealing.

First and formost let me impress on you the fact that vaccines have not superseded all other means of treatment; the general does not get rid of his infantry because of the accuracy of the fire of his artillery, nor has the torpedo superseded the big gun. There is just as much, nay, more, need of accurate diagnosis as there ever was. It is the only true basis of treatment. What we all need to learn—and it is only experience that can teach us—is when we may rely on our old pop-guns, and when the new weapons are to be called in to supplement them. If you have ever seen a case of cystitis treated by vaccines alone, and then had the advantage of seeing the same patient subjected to the influences of bed, diet, diluents and urinary antiseptics as well, any doubt which you may have of the value of these latter will soon vanish. Don't ignore them. They may effect a cure whilst you are waiting for your vaccine.

It is commonly supposed you must have the services of a trained pathologist if you are to get a satisfactory vaccine. Far be it from me to decry the trained pathologist. He is one of the greatest helps to us in our treatment if we use him aright. But you must take him into your confidence, confer

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with him over the case, let him see it from your point of view and try and look at it from his as well. But the highly-trained pathologist does not grow everywhere, and moreover he is an expensive luxury. I am anxious to point out to you that for vaccine purposes in particular it is possible to be content with a pathological dresser and yet get excellent results. That is what we have been doing with the results I have just laid before you, and I am sure our pathological dresser will not feel insulted when he knows I am not describing him as a trained pathologist.

The first thing to be done is to isolate the organism, and here some difficulty may be experienced, especially if—as is almost sure to be the case when the disease is of long standing—the infection is a mixed one. Having selected what you deem to be the dominant organism, it is grown usually on an agar medium. By means of a standard loop of platinum wire a known amount by weight or volume is scraped off the medium and placed in 4 c.c. of sterile normal salt solution. These loops can be bought ready made or are made in the laboratory. After being weighed empty and full on several occasions the operator will have no difficulty in collecting near enough for all practical purposes the amount that he requires, viz. 2 milligrammes. The tube which contains the 4 c.c. and the bacteria is pasteurised at a temperature of 65° C. for half an hour in order to kill the bacteria. The solution is now divided, say, into eight parts, each of which is placed in a small ampoulle such as I have in front of me. Each ampoulle, which of course contains 0·5 c.c. of the solution, is separately sealed, again heated, and then placed for twenty-four hours in an incubator to make sure that it is perfectly sterile. If no growth has taken place it is ready for use, and its contents can be injected into the patient.

It is hardly necessary to call attention to the need for perfect asepsis.

I have not mentioned the term "opsonin," which may possibly cause you some surprise, but I have done so advisedly. This is a clinical lecture. I know that the method which I have just described will not harm your patients; I am sure that in many instances it does them a great deal of good. I am equally sure from personal experience that it is advisable to administer a second dose some week or ten days later, and many more if the symptoms do not subside, but further than this I am not prepared to go. It would be foreign to my purpose to enter into a minute criticism of the opsonic theory and its fallacies. If you desire to learn what is to be said for and against it, let me commend to your notice a paper by Dr. Horder, which was read before the Abernethian Society last December and is now re-published in pamphlet form. I am sure you will derive much profit from its perusal.

*August 31st*, 1908.

# CASES IN THE OUT-PATIENT DEPARTMENT, ST. MARY'S HOSPITAL.

By WILFRED HARRIS, M.D., F.R.C.P.

CASE 1.—Here is a man, æt. 53 years, a hansom-cab driver, whose complaint is of swelling of his feet and legs and scrotum, and of shortness of breath. It is obvious as soon as he pulls up his trousers above his boots that the legs are much swollen, the skin is shiny and there is pitting on pressure. If this swelling and œdema were of one leg only we should at once think of some local cause such as thrombosis of the femoral vein, or phlebitis, but since the œdema is bilateral it must be due to some cause affecting the return of blood to the heart from the inferior vena cava, such as heart failure, or thrombosis, or mechanical pressure on the vena cava. There are, however, apparent exceptions to this rule, in commencing right ventricle failure, or renal disease, in a person who has previously suffered from femoral thrombosis on one side, or is hemiplegic. In these exceptions the œdema in the leg on the affected side may be very much greater than in the other, but even then there will be slight pitting on pressure over the crest of the tibia, which will be sufficient to show that the œdema is bilateral, and therefore probably due to heart failure or renal disease. One word of caution against a mistake that I have seen made more than once, and that is mistaking for œdema the slight depression that may be produced in the subcutaneous fat over the crest of the tibia. In our patient sitting here the clue to his trouble is at once afforded us by feeling his pulse, a point that should never be omitted from our routine, owing to the valuable information this simple test often yields. Notice how thick the artery wall is, and though moderate pressure comparatively easily obliterates the pulse-wave, yet the artery still feels almost as prominent and cord-like as before. If we were to take his blood-pressure now with the Riva-Rocci sphygmomanometer it would probably register no more than 130 or 140, though in cases with such an artery as this, before heart failure occurs it is not infrequent to obtain a reading of 250 or even 280 mm. of mercury. We will now

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send him to the inner room to pass a specimen of urine, and to strip to the waist. On examining his heart the first thing that strikes us is the large area of strong heaving pulsation which can be felt as far as the middle of the left axilla. Auscultation shows us a blowing systolic murmur audible from the left nipple to the mid-axillary line, while percussion and auscultation of the lungs show that there is nothing amiss there beyond a few *râles* due to congestion at the bases. We must not forget in such a case to examine the lungs, because we want to be sure that there is no disease in the chest, such as a pleuritic effusion, or bronchiectasis, to displace the heart so far over into the axilla. That being ascertained, it is clear that the heart must be enormously dilated, and the cause of the œdema of his legs is thus proved to be due to heart failure. Is this heart failure secondary to a valvular lesion, or to the high blood-pressure of arterio-sclerosis or chronic renal disease? He has never had rheumatism, and the strong heaving impulse of the left ventricle practically decides against a primary valvular lesion, even of aortic regurgitation, as in these cases when right heart failure and extensive œdema have supervened, the left ventricle impulse always loses most of its force. The condition here, then, is certainly dilatation of the excessively thick muscular left ventricle that is associated with arterio-sclerosis or contracted granular kidney, or with both diseases combined. In some cases the arterio-sclerosis is the primary lesion, in others the kidney cirrhosis is primary, but the eventual picture of thick arteries, hypertrophied left ventricle, and albuminuria is much the same in either case. The urine report here shows a specific gravity of 1010, with a well-marked trace of albumen, so in all probability in this case the development of contracted granular kidney has produced the arterio-sclerosis and high blood-pressure which has ultimately led to his present condition of heart failure. For *treatment*, rest in bed, and give large doses of digitalis, pushing the drug until diuresis is obtained. It is in cases of enormously dilated heart such as this, without sclerotic valvular disease, that we see the best results from digitalis.

Case 2.—Here is a girl, æt. only 19 years, and unmarried, whose complaint, like that of the man we saw just now, is of swelling of the legs which has been coming on for the past month. She is of good colour and does not appear ill, and her pulse, as we should expect at her age, is perfectly soft. Very occasionally we do find in young people a hard cord-like artery. This is either due to post-scarlatinal kidney—a condition precisely similar in its effects to contracted granular kidney—or else to an inherited tendency to arterio-sclerosis. This is very liable to run in families, the members of which usually suffer sooner or later from chronic Bright's disease, gout, diabetes, apoplexy, or angina pectoris. We will examine this girl's chest next, and we cannot help noticing the very large pendulous breasts, with large and very dark areolæ around the nipples. Her heart appears normal on auscultation, so we will next examine the abdomen, which appears full and prominent as she stands. Now as she lies on the couch it is at once evident that the girl is pregnant about eight months, as the uterus can be felt about half-way between the navel and the epigastrium. Note, too, its intermittent contractions, as the uterus hardens under the hand until it feels like a solid tumour. Note, too, the fluid thrill through the swelling. No placental souffle or fœtal heart sounds are audible in this patient, but the intermittent uterine contractions are positive evidence that we have here a case of pregnancy, which by pressure on the inferior vena cava has caused the enormous œdema of the legs. To make sure that this same pressure has not damaged the kidneys by obstructing the renal veins, producing albuminuria, we have asked the nurse to obtain a specimen of the urine, and the clerk has just reported that it is free from albumen. We may therefore hasten to assure the girl's mother, who appears rather anxious about her, that her daughter will most certainly be delivered of the cause of her trouble within the next few weeks.

Case 3.—We may say that it never rains but it pours! Here is another young girl, æt. only 20 years, also unmarried, who is complaining of vague pains in her back and of swelling of her stomach, with nausea. On examining her abdomen the cause of the swelling is seen to be again a physiological one, as the uterus can be felt more than half way between the pubes and umbilicus, and a well-marked placental souffle can be heard on the right side. She now tells us that her periods have

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been absent for the past four months. Note the soft feeling of the tumour, and that it is a little difficult to make out exactly the upper edge of the swelling. Remember it is quite easy to mistake a dilated bladder for pregnancy at the fourth or fifth month. I remember, when I was Casualty Physician some years ago, a woman bringing her daughter on account of the cessation of her periods for the past five months. The girl did not appear anæmic, and suspecting therefore a physiological cause I examined the abdomen and found a tense swelling nearly up to the navel. Feeling satisfied as to the cause, I proceeded to inform the girl that she was pregnant and that she ought to have known it, a statement which was received with no apparent surprise by the girl, but with loud lamentations by the mother. I then listened to the girl's heart and found she had well-marked mitral stenosis, a quite sufficient cause for her amenorrhœa, and so, feeling a little doubtful, I told the nurse to pass a catheter. This was done at once, sixty-eight ounces of urine were drawn off, and the abdominal swelling completely disappeared. She then admitted that she had not passed her urine at all that day. I feel I ought to have apologised to the girl, but instead I admonished her that although she had got off this time, she must not expect to get cured so easily another time. Both she and her mother were profuse in their thanks, and I feel certain that they both went away convinced that the girl owed her deliverance to me.

Case 4.—This young girl, æt. 12 years, it appears attended here last week while I was taking the wards, and the notes say that there are numerous patches of purpura on the legs, with periosteal swellings. She is clearly anæmic, and her mother has just told us that so long as she lies up and keeps off her feet her legs are all right, but to-day the lumps have come out again as soon as she got up to come to the hospital. Now look at her legs and notice the bruised appearance around the ankles, due to fading hæmorrhages in the skin, or patches of purpura. Scattered all over the fronts of the legs, nearly as high as the knees, are numerous irregular wheals, some circular, others sinuous in outline, whitish in colour along the summit of the ridge, the rest of the skin appearing somewhat congested and swollen. Her mother tells us that with the eruption of these patches the legs become irritable and somewhat painful, and that after some hours the bright red hæmorrhages appear in their place. Passing my finger along the crest of the tibia I can feel no "periosteal swellings" as described in the notes, and I shrewdly suspect that similar wheals on the skin were mistaken for them. We have then, here, purpura associated with urticaria and pain in the limbs, and the distribution of the purpura, as you can see from the fading stains, is mostly around the ankle-joints. This disease is described sometimes as "rheumatic purpura," "Schönlein's disease," or "peliosis rheumatica." It affects children mostly, and its association with rheumatism is often not very evident beyond the occurrence of joint pains. Endocarditis has, however, been found occasionally associated with it, though there is no sign of its presence in our patient here. Moreover, salicylates do not seem to do any good in this disease, though we must not therefore deduce the inference that the disease is not rheumatic, because in chorea, whose very close association with rheumatism is undoubted, salicylates are similarly of little or no benefit. The disease is apt to be troublesome often for several weeks, the wheals and purpura recurring as soon as the child is allowed up, though nothing may appear amiss so long as the patient is kept in bed. A closely allied variety of this disease is what is known as "Henoch's purpura," in which in addition to the urticaria and purpuric patches on the skin there are occasional attacks of severe colic with hæmorrhage from the bowel, and perhaps hæmatemesis. In these cases it is highly probable that a similar urticarial process is affecting the gastro-intestinal mucous membrane, and the same point is noticeable in them also, that the child keeps well as long as he or she is kept in bed, though within an hour or two of getting up the wheals and itching and hæmorrhagic spots begin to appear. The tendency to urticaria and purpura, especially when the patient is up and about, might suggest an abnormal weakness of the capillary walls, or an excessive tendency to escape of serum and even red blood-cells through the capillary walls, owing to deficiency of the blood in calcium salts; I have not, however, found treatment with calcium lactate produce any improvement, though I have seen improvement follow ten-minim doses of oil of turpentine, as recommended by Sir Stephen Mackenzie. Remember to avoid

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giving iodide of potassium in purpura. Iodides sometimes cause most severe purpura, and may increase an existing purpura.

CASE 5.—The next patient who presents any points of special interest is this young married woman, æt. 23 years, who tells us in husky tones that her voice has been gone for the last four months, and that she has been having pain and difficulty in swallowing for the past month. Looking at her tonsils and pharynx you can see that there is nothing there to account for her trouble, so the mischief must be below, in the larynx and epiglottis. Her symptom of pain on swallowing is very suggestive of inflammation or ulceration of the epiglottis, and this, in association with huskiness of her voice for so long as four months, makes me think of tubercular laryngitis. She does not look anæmic, however, nor wasted, and she says that she was never previously troubled with cough. Tubercular laryngitis is very rarely primary, and is nearly always a secondary infection from tubercular sputum in advanced phthisis; in the later stages ulceration of the epiglottis is not uncommon, giving rise to severe pain on swallowing, and in some patients tubercular ulcers are found on the back wall of the pharynx, on the tonsil, tongue and cheek. We will now examine the larynx itself with the laryngoscope. Notice what a brilliant light our new Nernst filament gives in comparison with the older form of electric lamp; with the laryngeal mirror we can see, after some little trouble, that the vocal cords are intensely congested and red, but there are no signs of ulceration visible. The region of the arytenoids and ary-epiglottidean folds also appears swollen and congested, and this general marked hyperæmia is unlike the condition usually met with in tubercular laryngitis. In the latter, although the cords are somewhat pink and have lost their normal brilliant white appearance, the general tone of the larynx is rather anæmic. This patient's complexion is ruddy rather than pale, nor is she wasted, and she says she was never previously troubled with cough. She goes on to state that she has never been well since she had a still-born baby of seven and a half months just after last Christmas, and with this remark of hers we have at once the key of the puzzle. Syphilitic laryngitis is an occasional manifestation of the secondary stage, though it is far less common than the well-known sore throat from ulcerative tonsillitis. She still has some patches of secondary syphilitic eruption upon the flexor surface of the right forearm, and she she says that for four months before the baby was born the rash was much more extensive on her chest and other limbs, though the right forearm then escaped. The diagnosis of secondary syphilis is therefore certain, and we will treat her with the biniodide of mercury.

CASE 6. *Gummatous neuritis of lumbo-sacral plexus.*—This man is a police-sergeant, and his condition is one that is most important to recognise, because it is rapidly curable if properly treated in time. He was sent to me as a case of peripheral neuritis, and you can see that as he walks he drags his left foot and raises the left knee high so that the dropped left foot shall clear the ground, the left foot flapping as he walks. The right leg moves quite well, and he says there is nothing the matter with that. According to his statement, a month ago the left leg began to get weak, and rapidly got worse, causing this difficulty in walking. He says there is no pain and very little numbness. All the muscles in the right leg and foot are normal, and the sphincters are quite unaffected, but the paralysis of all the muscles of the left leg below the knee is complete, and there is also distinct weakness of the left hamstrings. The left knee-jerk is slightly diminished, and the left ankle-jerk absent. There is clearly some wasting of the muscles of the calf and leg, and the electrical reactions, which have been carefully taken, show complete reaction of degeneration in all the muscles below the left knee, with some diminution to faradism in the hamstrings and quadriceps also. This rapid onset of weakness and wasting in the muscles of one lower extremity is very characteristic of a gummatous neuritis of the lumbo-sacral plexus, a condition which I have seen several times, although the books make little or no mention of this condition. In my experience it is a far commoner condition in the lumbo-sacral region than in the cervico-brachial. The lesion is not in the spinal canal, as is shown by the unilateral symptoms, and the absence of sphincter involvement or pressure paraplegia. Gummata in the spinal canal, pressing on the spinal cord, do, of course, occur, but the symptoms are then more

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or less bilateral. In these cases of gummatous neuritis of the plexus the lesion is probably around the exit of the nerves from the intervertebral foramina, and the initial symptom is always pain, usually of some severity and lasting some weeks. This man did not make any complaint of pain, but on cross-questioning him he now states that before the leg became weak he had sharp "rheumatism" pains around the left hip for a fortnight, and that when these pains ceased the leg became weak. He has now been taking biniodide of mercury for two weeks, and is also being treated with galvanism to the wasted muscles, and he has already begun to improve, and feels his foot is stronger. This particular case is fairly easy to diagnose, because his legs are much scarred with syphilis, and he readily admits having suffered from the disease eight years ago. The prognosis here is very good, though as the wasting has gone to the stage of full reaction of degeneration it must be many months before the muscles regain their full power. If the disease is not recognised in time, and the proper treatment applied, the paralysis becomes permanent owing to the destruction of the nerves with scar tissue.

*August 31st*, 1908.

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We have received from Messrs. John Bale, Sons, and Danielsson a copy of 'Sprue, and its Treatment,' by W. Carnegie Brown, M.D., M.R.C.P. The author makes a special endeavour to emphasise the necessity of early treatment, and he describes the various methods which are now employed. As he very rightly observes in his preface, the question in the tropics is not, "What treatment is best?" but rather, "What treatment is available, and how far can the means at one's disposal be adapted to the case in hand?" For this purpose the book is admirably written, and is rendered extremely valuable by attention to minuteness of detail so essential to lucidity and practical utility. The volume contains an excellently-written chapter on the hygiene and prevention of sprue, in which Dr. Carnegie Brown aptly and pithily points out how true prophylaxis against sprue depends on intelligent and not over-specialised attention to the general rules of health in the tropics. It is an interesting fact that so far as sprue is concerned the spread of the disease has coincided with a great reduction in the quantity of meat consumed by Europeans, and the author shows that in the tropics a certain quantity of animal food is essential to health, experience leading to the belief that vegetarianism conduces to the general condition in which sprue originates.

## THE SYMPTOMATOLOGY, DIAGNOSIS, AND TREATMENT OF SIMPLE CHRONIC AND TUBERCULOUS PROSTATITIS.

By CHARLES GREENE CUMSTON, M.D.,
Boston.

Simple chronic prostatitis and tuberculous prostatitis develop in about the same conditions. Both affections belong to young or middle-aged people, and quite exceptional before puberty and very rare in elderly subjects; it reaches its maximum frequency between twenty and forty years of age, at the epoch of sexual activity, and this in spite of the influence recently supposed to exist of prostatic hypertrophy on the development of prostatitis.

However, several years ago I operated on a man æt. 80 odd years, at the Maine General Hospital in Portland, who had suffered from all the symptoms of an ordinary hypertrophy of the prostate, but which, when the gland was exposed through a perinæal incision, proved to be an enormously enlarged prostate due to tuberculosis. Upon section the gland was riddled with tubercles and so soft that it could only be removed piecemeal with the finger.

In 26 cases observed by Socin 13 patients were 30 years of age, while out of 60 cases Simmonds found the disease more frequent between the ages of 20 and 50 years; one of his patients was 18 months old and another 7 years old; 15 were in the thirties, 15 in the forties, 16 in the fifties, 6 in the sixties, 7 in the seventies and one in the eighties.

Gonorrhœa is the most usual ætiological factor of prostatitis, and although we all know of undoubted cases of chronic prostatitis in subjects who have never had gonorrhœa, and likewise tuberculous lesions of this gland in subjects who have always had a clean urethra, it nevertheless remains a fact that gonorrhœal urethritis is often complicated with a prostatitis, as many statistics go to show. Mortagnon found prostatitis in 70 per cent. of cases of urethritis, Pozzoli in 80 per cent., Segond in 40 per cent., while Goldberg found it in from one third to one half of his cases. The infection of the gland by tuberculosis takes place, probably, in a large majority of cases by way of the blood or the lymphatics. Primary infection coming directly by way of the urethra has been

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believed possible by Verneuil, Cohnheim and Fernet, and these writers admit that coitus with a woman having a genital tuberculosis may infect the anterior urethra, and that from here the bacilli reach the prostate. In a recent case recorded by Guépin he incriminates buccal coitus. Von Frisch has incriminated soiled catheters and the wearing of clothes infected with tuberculous expectoration. Certain recorded facts would seem to demonstrate that there is a possibility of such infection, but they must be very rare indeed.

Although chronic prostatitis arising from rheumatism or arthritism has been upheld, I am of the opinion that heredity plays a very important part in the development of tuberculosis of the prostate, and out of thirty-five cases observed by Desnos sixteen gave an hereditary tubercular history. In the few cases that have come under my observation I found that this was also the case. Certain authors have believed that chronic prostatitis and the tuberculous form could be brought on by excessive coitus, masturbation, stricture, riding horseback or the bicycle, but these, I believe, are simply accessory causes merely producing prostatic congestion.

I now come to the symptomatology, and will first take up that of chronic prostatitis. As is well known, this organ is composed of a glandular and conjunctive tissue, and muscle-fibres, so that the simplest and most rational classification is that of catarrhal parenchymatous prostatitis and interstitial prostatitis.

In the parenchymatous type we have as functional symptoms the disturbances in sensation, usually composed of an ill-defined fullness in the perinæal region, while occasionally there is an indefinitely localised pain, which becomes increased when the patient has been in a sitting posture for a long time, and, like the pain of calculus, riding in a carriage and walking produces it. It sometimes extends to the sacral region and the patients complain of a burning sensation.

The genital disturbances are very variable, and although without doubt they truly exist they are frequently exaggerated by the patient. Sexual desire is often quite pronounced in the beginning of the affection, but diminishes and may finally disappear. Erection becomes incomplete, but it is rarely absent. Ejaculation occurs quickly and is sometimes painful, and not infrequently the patients complain of sharp pain after the act. As to hæmospermia, Guyon believes that it is due to an inflammation of the vesicles, but, of course, this does not exclude a prostatitis, and Keersmacker has met with it independently of any lesion of the seminal vesicles.

Nervous disturbances appear in the form of a loss of appetite, pain in the spine, loss of memory, or a more or less marked depression of the intellect

The disturbances of micturition consist of tenesmus and frequency. The repetition of the act may be marked in some cases during the day when the patients are moving about, or, on the other hand, during the night, when the prostate becomes congested from the horizontal position. In some cases the desire to urinate increases at certain definite times in the day, usually towards the evening or the first thing in the morning. In others, after micturition a persistent contraction of the sphincter gives rise to a very disagreeable sensation in the perinæum.

The urine washes down white shreds, which are generally elongated or somewhat rolled up, these being met with during the first part of micturition and also in the last drops expelled. Clark has mentioned the presence of casts similar to those coming from the kidney, but taking on the shape of bottles or pears. Contrary to the assertion made by Ultzmann, albumen is not present. The urethral discharge affects the patient mentally. Defæcation will bring a thick white or yellowish-white liquid to the meatus, having a spermatic odour and an alkaline reaction. The passage of the fæcal mass acts like massage by direct pressure on the prostate, while contraction of the prostatic sphincter acts in the same way by expressing the contents of the gland. This liquid, which may also appear intermittently and spontaneously, in the form of small aborted ejaculation, causes staining of the linen, the spots being quite large with irregular borders. Microscopically one finds numerous cylindrical cells, often united in masses, presenting round cells at their base, and sometimes even complete casts of the excretory ducts, numerous leucocytes and Boettcher's crystals.

Catheterisation shows general sensitiveness in the prostatic region and the olive of the explorer will sometimes bring back muco-pus, which in other cases only makes its appearance after the instrument is withdrawn. A slight retention may also be

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observed. With the endoscope the verumontanum will be found enlarged, very red, and hyperæmic. The orifices of the ejaculating canals project slightly, while a more or less marked redness is seen throughout the entire mucosa, the latter often being swollen in certain points. Rectal examination, although disagreeable to the patient, does not produce any severe pain, and when the prostate is massaged one may often express a certain amount of liquid, which will make its exit at the meatus.

The products of the physiologic secretion are increased in amount and are mixed with catarrhal or purulent pathologic elements. The lumen of the excretory canals is diminished or obliterated on account of the inflammatory process in the walls. The secretions stagnate and from this results an increase in the size of the prostate, either in one or both lobes. The organ becomes more and more distended, and when examined *per rectum* it is found rather soft, and to the feel is very much like a rubber ball. Its surface is unequal and ill-defined lumps may be felt over its surface. The vesicles are sometimes indurated.

Referring now to the interstitial type of prostatitis, I would say that the functional symptoms differ little from those encountered in the parenchymatous form, but they are very much less marked. The patient will only complain of an indefinite pain and sensation of weight in the perinæum. There is only a slight amount of discharge, just enough to stick the lips of the meatus together. The urine contains white shreds, but these are not so long and are fewer in number than in catarrhal prostatitis.

In this form of prostatic inflammation the connective-tissue elements are those involved; the infiltration occurs around the glands, becomes organised, and transforms into a very dense tissue, which does not permit of dilatation. Consequently *per rectum* one finds the gland hard to the feel, rather painful on pressure, and irregularly increased in size, so that it appears asymmetrical. Its surface is covered with small rounded lumps, whose contour is poorly limited. These bosses are to be found especially on the periphery and external borders of the gland. They are placed, so to say, in straight lines, so that they give the gland rather a square shape, and it appears as if surrounded by a frame, this being all the more accentuated the greater the central depression.

Tuberculous prostatitis takes on many forms, but I believe that one may divide it into two types, according to the predominance of the lesions in the urethral mucosa, or in the glandular parenchyma.

The functional symptoms of the mucous membrane type are those of a deep urethritis. In the beginning they are not marked, but increase as time goes on, although their severity is in no way in relation to the minute lesions that exist. Disturbances of the micturition represent the first act of the drama. The patient has frequent, painful, and imperious desire to urinate, this occasionally ending in a pseudo-incontinence. These symptoms, I believe, indicate an extension of the process to the bladder, but this is not always a certain indication, because they are encountered in cases where the bladder is perfectly free of disease.

Evacuation of the bladder is slow, the stream of urine is delayed, the patients feel as if they had an incomplete micturition, and they suffer from tenesmus. Not infrequently there is partial retention, and later on in the process, when the infiltration has involved the vesical sphincter, this muscle becomes paretic and the urine escapes drop by drop. Consequently a true incontinence develops.

Hæmaturia is met with in all the stages and usually appears early in the process, and can be rightly compared to the premonitory hæmoptysis of pulmonary phthisis. In this case it occurs without any ulcerative process being present and is merely due to the hyperæmia. The blood comes away, like the pus, with the first drops of urine voided, but it is more usually met with at the end of micturition. The urethral discharge is very similar to that met with in gonorrhœa. Without any inflammatory phenomena the mucosa of the prostatic urethra is alone involved and gives rise to the discharge, and we have here what Ricord used to call tuberculous blennorrhœa. Microscopically the bacillus of tuberculosis may be found, and not infrequently during the very early stages of the affection.

Catheterism, besides being usually difficult on account of the urethral spasm, is occasionally extremely painful and throws little light on the condition of affairs. An endoscopic examination is of little use, and I believe should be proscribed. Rectal palpation gives little information, especially in the beginning of the disease. The prostate will

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probably not be found enlarged, while its surface is smooth and it is normal in consistency. However, massage of the gland will express prostatic secretions which, collected at the meatus, will be found composed of pus, in which, as I have already pointed out, the bacillus of tuberculosis may be often found. In more advanced cases the lesions are no longer limited to the urethra, and tuberculous foci develop more deeply and finally may be detected by the finger in the rectum.

The functional symptoms of parenchymatous tuberculous prostatitis may be absolutely wanting, but when the disease develops in the peripheral layers of the gland, thus being superficial and near the rectum, the symptoms are represented by some weight in the perinæum and a disagreeable sensation during defæcation. The pain increases in the sitting posture and may also be produced by walking or the jolting of a carriage; they are quite independent of micturition, the disturbances of which are only slightly marked in the beginning of the process.

A purulent deposit, which has accumulated in the prostatic urethra when suppuration is sufficiently marked, is washed down with the first drops of urine, while the urine in the middle of micturition is always clear. The presence of pus at the end of micturition is a symptom of cystitis, but when the glands are gorged with pus the contraction of the peri-prostatic muscles may cause it to appear at the end of micturition.

The discharge in the parenchymatous form is purulent, and occurs during defæcation or slight massage of the prostate. Chronic constipation is frequent in tuberculous prostatitis.

When the urethral discharge is the result of small tuberculous abscesses which have opened into the urethra it occurs during the interval of micturition, while in the more advanced stages of the disease the suppuration is almost continuous. I would point out that the extent of the focus of disease must not be judged by the quantity of discharge, because certain old cavities which have become organised give rise to little secretion, while a young focus undergoing disorganisation will suppurate abundantly.

Rectal examination will throw much light on the diagnosis. The prostate will be very sensitive, and sharp pain on pressure is an early symptom and very useful from a diagnostic point of view. The gland will be found increased in size, but rarely is this increase uniform, and usually one of the lobes is larger than the other. More or less distinct bosses can be detected over its surface, and in those instances where the tubercle is very superficial the prostate feels as if studded with shot. The tuberculous lesion has a tendency to become isolated, and as long as it does not give rise to suppuration the finger can usually limit its contours.

The tumefaction of the prostate rarely reaches any marked degree, but when, as in some cases, it is double its size, it usually contains one or several purulent foci. Fluctuation is rarely met with, for the simple reason that the walls of the abscess undergo an ulcerative process early in the disease, so that a cavity with irregular walls is formed. The seminal vesicles are involved at the same time, or before the prostate is diseased. Sensitiveness and induration indicate their participation in the process.

From what has been said of the various manifestations of chronic prostatitides and tuberculous prostatitides, we should endeavour to ascertain what differential value may be attributed to them, which of these symptoms will allow one to make a diagnosis, and when they are wanting what little symptoms will aid us in suspecting the transformation of a simple chronic inflammatory process into a confirmed tuberculous one. It is quite evident that the genital or sensory disturbances throw little light on the matter, and besides, that their intensity varies according to whether one is dealing with the parenchymatous or interstitial type, with the mucous or parenchymatous tuberculous forms; they are merely a feeling of weight in the perinæum and a disagreeable sensation during defæcation, this being met with in the interstitial form and the parenchymatous type of tuberculous prostatitis. And what is still more, the distinct lesions felt *per rectum* do not give rise to any of these symptoms, and, on the other hand, the latter may exist without any morbid process being present.

The disturbances in micturition, although giving no precise indications, occasionally allow one to suspect the condition of affairs. Absent, or only slightly marked in chronic prostatitis—unless there is a cystitis, in which case it is relatively easy to distinguish the vesical phenomena—they, on the contrary, indicate the commencement of the phy-

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matosis. The desire to pass water becomes both imperious and frequent, quite independently of any extension of the process to the bladder and of any neuropathic influence. Incomplete retention, although sometimes observed in chronic prostatitis, is common and occurs frequently very early in tuberculous prostatis.

The urethral discharge offers little diagnostic certitude. Present as only a slight discharge in the interstitial type, it is completely wanting when the specific inflammation remains peri-glandular, and this condition may remain as such for a long time. But when it exists, its character being distinctly purulent, its emission never spontaneous, the necessity of pressure either with the finger or fæces in order to produce it indicate that it is probably of a tuberculous nature. Spontaneous hæmaturia, occurring both during the day or night, uninfluenced by movement and not stopping when the patient rests, suddenly ceasing, represented by a few drops of blood at the commencement of urination, but more frequently at the end of the act, is in favour of a tuberculous process in the prostate, particularly in the mucous type.

Catheterism indicates nothing conclusive, since in both cases it causes a severe pain in the prostatic region. I believe that little value can be given to an endoscopic examination in the cases we are considering.

Rectal examination is more useful and the results more positive, and in many cases it is the only diagnostic source upon which we can base our conclusions. It should always be performed with gentleness, because, when the finger suddenly passes through the anal sphincter it causes pain to the patient, which might be the cause of very regrettable mistakes.

Chronic prostatitis and tuberculous prostatitis finally end in producing an asymmetry of the gland, since the increase in volume is usually met with in only one of the lobes. However, in the simple chronic type the gland has a more uniform and softer consistency, while in the interstitial type its surface is regular, the bosses are larger, less isolated, and sunk in the parenchyma of the gland, so that a diagnosis can be made. Involvement of the seminal vesicles is of great importance, and when they are very sensitive and present irregularities over their surface the diagnosis of tuberculosis may be made with a considerable degree of certainty. In chronic prostatitis they occasionally increase in size, but their surface is smooth.

Bacteriological examination is, of course, very important. Microscopical examination of the liquid expressed by massage, and which should be collected after irrigation of the anterior urethra has been carefully done, will usually reveal the presence of the specific bacillus. This, however, is not always the case, because it is more particularly met with in the early stage of the mucous form of tuberculous prostatitis. Culture and inoculation may sometimes remain sterile in results, and nevertheless the evolution of the prostatitis is rapid and distinctly tuberculous. Such facts prove that when the bacteriologic result is positive the diagnosis is evident, but that when it is negative. the tuberculous nature of the lesion cannot be eliminated, and one should still cling to the clinical phenomena in the diagnosis. If at an advanced period, when the lesions involve the bladder, seminal vesicles, the spermatic cord, epididymis, without speaking of the possible localisation in other organs the diagnosis of prostatic tuberculosis may not be made, and at this period no hope of a cure can be entertained because it is too late. On the contrary, in the period where one finds few of the classical symptoms it would be an easier matter to deal with the affection because surgical interference might result in a cure. It is for this reason that I particularly emphasise the necessity of an early diagnosis, and, in order to facilitate this I would refer to two little symptoms which are observed in tuberculosis of the prostate. The first is the appearance of light pellicles floating on the surface of the urine—an indication of an extremely mild type of desquamation. The other is atrophy of the testicle, without any induration or nodules in the epididymis. In calling attention to these two symptoms I do not wish to convey the idea that they are to be always found, because this is not the case; but when they do exist they are certainly of great diagnostic value.

Simple chronic prostatitis and tuberculous prostatitis are very similar in their development. This is essentially irregular, occurring in congestive and inflammatory attacks, which result in a recrudescence of the symptoms. Both forms are very tenacious, because, even in the simpler varieties, the most varied treatment will have no effect, while in the tubercular variety its development is slow

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and silent when the tubercles develop excentrically; on the contrary rapid and with marked symptoms when the prostatic mucosa is involved.

The prognosis of the various forms of prostatitis differs. In chronic prostatitis it is never serious, because the lesions met with finally end by disappearing, while in tuberculous prostatitis it should be most guarded, although it need not of necessity be absolutely bad, because—and I repeat this again—regression of the specific products is possible, especially if, on account of an early diagnosis, proper therapeutic measures have been instituted and have modified the developing lesions. I have seen a cure in one or two cases, at least an apparent cure, because the patients have remained well for several years without any symptoms which would cause one to believe that their former affection had ceased to exist.

On the other hand, the prognosis will vary greatly according to the general condition of the patient, and whether or not he has other tubercular foci in the lungs or elsewhere. When these do exist the outlook, of course, is not favourable. The more or less rapid evolution of the disease and its tendency to invade the upper urinary tract must also be considered. The participation of the testicle, the vas deferens, the seminal vesicles, and peri-prostatic tissue darken the prognosis quite as much as when the bladder itself is involved.

A complete treatment should be prophylactic, ætiologic, symptomatic or local. Prophylaxis of tuberculous prostatitis certainly exists to a certain extent, inasmuch as subjects predisposed to tubercular lesions should be informed of the danger of gonorrhœal infection, or when they are subjects of old chronic urethritis this lesion should be carefully treated until absolutely cured. These subjects should be told to eat well, to avoid all excesses, and to exercise sufficiently without becoming tired. Horseback riding I believe to be pernicious. Douches, dry friction, sea baths, and a life in the open, particularly in mountainous countries, are to be advised. General treatment is also of great importance here, as in tuberculosis of any other organ, and, although I do not profess to be an expert prescriber, I shall take the liberty of giving a few formulæ which have rendered me good service.

In the first place, and foremost of all, stands cod-liver oil, and the phosphates and the glycero-phosphates seem to me to have their indication as general tonics. The two following prescriptions will be found useful:

℞ Sodium phosphate . . 6·0
Potassium phosphate . . 3·0
Vini hispani . . . 200·0
Syr. cort. aurant. . . 50·0
*M. D. S.* A liqueur glass after each meal.

℞ Calcium glycero-phosphate . 0·30
Magnesium glycero-phosphate 0·10
Ferrum glycero-phosphate . 0·05
Pepsin aseptic . . . 0·15
Pancreatin . . . . 0·05
In chart. No. 1.
*S.* One powder after breakfast and dinner.

Fowler's solution and, in fact, arsenic in all forms may be employed. Sodium cacodylate at the dose of five centigrammes a day has, I believe, been successfully used in some cases. I have given arsenic as follows.

℞ Sodium arseniate . . 0·05
Vini cinchonæ . . . 50·0
Glycerine . . . . 50·0
Syr. cort. aurant., *q. s. ad.* . 300·0
*M. D. S.* A dessert-spoonful with each meal.

Tannin, iodoform, eucalyptol, guaiacol, and creasote can be given in the the form of pills, cachets, suppositories, or rectal injection. The following combination has certain therapeutic virtues.

℞ Terpin. hydrate . . . 0·05
Guaiacol . . . . 0·04
Sodium benzoate . . 0·03
Ext. cinchonæ . . . 0·02
Excipient *q. s. f.* pil. No. i.
*S.* 5–10 pills a day.
Creasote . . . . 10·0
Sodium phosphate . . 40·0
D. in cachet No. xl.
*S.* 1 during each meal.

Medication applied topically *per rectum*, in the form of very hot irrigation, and also urethral irrigation, acts like a douche, but the indication for intra-urethral irrigation is not very precise. Generally speaking, it should not be employed as long as there is any retention or a prostatic urethritis. It necessitates manœuvres which may cause ulceration of the thinned mucosa covered with tuberculous lesions. When a prostatic urethritis exists, and especially when very painful,

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good results may be obtained by urethro-vesical irrigations without a catheter. The patient should first empty his bladder and then the meatus and glans are carefully cleansed. Then enough liquid to fill the bladder is injected and the patient should then expel it by micturition. A syringe should never be used. The solutions employed may be a 5 per cent. boracic acid or lactic acid; formalin at 2 : 1000, or a weak solution of tannin, or potassium permanganate. However, the treatment of choice is the use of concentrated solutions in the form of instillation given every second day. In my opinion silver nitrate is extremely dangerous in these cases, producing a discharge, hematuria and necrosis of the mucosa. Protargol, which has been proved in chronic prostatitis, is only of value in the tuberculous form when there is a mixed infection, in which case it diminishes the pain, the shreds decrease in number, but the specific lesion is unfavourably influenced by this agent. The action of iodoform is doubtful, although it has been highly lauded. Lactic acid has been used, but I cannot speak of it from personal observation. The following formula has been given by Witzach:

| | | |
|---|---|---|
| ℞ | Cocaine lactate . . . | 2·0 |
| | Acid, lactic . . . . | 10·0 |
| | Sterilised water . . . | 10·0 |

Ichthyol in a 2 to 5 per cent. solution has been greatly praised by Noguès.

The virtues of picric acid are interesting to know; not only is this agent painless, but it is also endowed with undoubted analgesic properties. As it is no way caustic it has, over sublimate, the great advantage of not producing irritation. It is an excellent antiseptic and possesses a keratoplastic power equal to that of pyrogallic acid. The only inconvenience in using this drug is the stain produced on the fingers or the patient's linen, but this can be removed by washing. To remove it from the skin a saturated solution of lithine carbonate, or water, to which potash or ammonia has been added, is all that is required.

As to the technique it is as follows: After the bladder has been emptied from 1 to 3 c.c. of a 2 to 3 per cent. solution is instilled in the membranous urethra. This instillation causes practically no pain if one is careful not to allow the solution to escape in the anterior urethra. The benefit is especially shown by an improvement in the secretions, which diminish as well as the painful symptoms. The frequency of micturition disappears and hæmaturia is not a contra-indication to this treatment, because it also usually ceases along with the other symptoms, and I am unaware that the drug has ever increased or started up the symptoms already present. An improvement in the patient is occasionally observed after a few instillations, but generally a number of weeks are necessary before one is able to judge of the results.

It is evident that some patients support intra-urethral treatment badly, and the effect of the solution on the mucosa varies very greatly from one case to another, so that the surgeon is frequently obliged to change the strength of the solution, also the medicament.

Massage of the prostate is the best treatment in simple chronic prostatitis, because it expels the secretions which have accumulated in the glands, while, on the other hand, it excites the activity of the peri-prostatic venous plexus and hastens absorption, but, in the tuberculous variety, the results obtained have not been so favourable. I do not think it can be absolutely proscribed in these cases, but, if resorted to, it must be done with great delicacy.

Surgical treatment has unfortunately few indications in the mixed prostatitides; however, when the diagnosis has been made in the very early stages of the disease, or when the tubercular lesions involve only the prostate and seminal vesicles, if the bladder is not involved and the patient's general health is good, prostatectomy may be proper and is the only means of preventing the process from extending. It would appear to me that, in this case, the perinæal route should be the one of choice.

*August 31st*, 1908.

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We have received from Messrs. Longmans, Green and Co. a copy of Vol. I, No. 9 of the 'Proceedings of the Royal Society of Medicine.' In the Dermatological Section, Dr. A. Whitfield's contribution entitled "Specimens from a Case of Syphilis" is of exceptional interest, and Sir Felix Semon's communication on a case of hysteria with very unusual laryngeal manifestations is well described by the President of the Laryngological Section as "remarkable in many ways." In a volume of this kind the degree of excellence must be expected to vary, but there are not a few who would question the following statement in a paper on cases of ununited fracture: "Cases of delayed union can generally be *easily* made to unite by hammering the region of fracture."

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# THE CLINICAL JOURNAL,

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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# A RESUMÉ OF RECENT CLINICAL STUDIES OF ATHEROMA AND ARTERIO-SCLEROSIS.*

By GEORGE OLIVER, M.D., F.R.C.P.Lond.

IN the hour at my disposal it is not possible to handle, however briefly, most sides of the study of atheroma and arterio-sclerosis. I shall therefore confine my remarks to three aspects of it; namely, (1) some general pathological data, (2) the arterial blood-pressure, and (3) some ætiological factors.

### (1) GENERAL PATHOLOGICAL DATA.

First of all I shall recall to your minds some anatomical facts, which you should keep in view while studying the diseases which degenerate and thicken the arterial wall.

#### *Some Anatomical Data.*

The accepted division of the arterial wall into three coats—adventitia, media and intima — is, strictly speaking, schematic, for the coats merge into each other. Therefore an artery may be broadly defined as a fibro-elastic tube, for the most part enclosing circular muscular fibres in the central part of its walls. The elastic fibres are more particularly distributed in the fibrous tissues of the outer coat (adventitia) and of the inner coat (intima), and preponderate in the central vessels; whereas the muscular tissue is limited to the middle coat (media), and is relatively to the other tissues more developed towards the periphery of the arterial system. This arrangement of the muscular and elastic tissues serves the important purpose of maintaining a certain head of pressure in the arteries between the systolic contractions of the ventricle, the higher pressure induced by the

* A Lecture delivered at the Medical Graduates' College and Polyclinic, London.

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latter being largely sustained by the elastic recoil of the large arteries, which acts in the manner of a supplementary heart while the heart muscle itself is resting. It is also important to remember that the arterial wall—at any rate the outer and middle coats of it—is furnished with a complete circulatory system of its own, consisting of arteries, capillaries, lymph spaces and veins. The inner coat, like the cornea, is, however, destitute of blood-vessels; but its nutrition may nevertheless be somewhat provided for by the fenestrated membrane of Henle, through the fenestræ of which the lymph circulating in the media may pass into the inner part of the intima.*

But notwithstanding all this provision for the nutrition of the wall of the artery, one cannot help entertaining a suspicion that the weak point in the anatomical distribution of the vasa vasorum is the scanty supply of vessels to the middle coat—and especially to its inner portion—and the absence of them from the intima. Surely this limited vascular supply may readily become contributory to degenerative changes in the middle and inner coats—the seat of arterio-sclerosis and atheroma. The circulation of blood and lymph in the arterial wall is of course a matter of prime importance in maintaining the nutrition of all the coats of the vessel; it is therefore easy to conceive how degeneration may be induced by persistent alterations in the physiological play of the functioning tissues—such as undue stretching of the fibro-elastic tissue in the large arteries and persistent muscular contraction in the medium-sized peripheral vessels. Besides this, should the arterial wall become distended with diminished movement in consequence of a rise in the blood-pressure, the lymph supply to the wall of the artery derived from the lymph spaces of the fibrous sheath around the vessel is diminished. Sir Lauder Brunton has pointed this out, and I cannot do better than quote his own words: "Each time that the artery is dilated by the blood forced into it during the cardiac systole the lymph is driven out of the sheath, while at the contraction of the artery (its systole) during the cardiac diastole, more fluid tends to flow in from the vasa vasorum. It is evident that if the difference between the size of the artery at each pulse and at each interval is great, there will be a correspondingly free circulation of lymph in the sheath of the vessel, but if the difference be very small the movement of the lymph will be slow and imperfect. Now with the higher blood-pressure the oscillation of the vessel is greatly diminished."*

There is another anatomical fact which has also a bearing on the clinical study of atheroma and arterio-sclerosis, and that is, the artery supplying certain organs (*e. g.* brain, spleen, liver, kidneys) remains large or undivided until it terminates in numerous divisions close to its destination. Such arteries distributed to important organs and derived from the central arterial system should be regarded as reservoirs of high pressure, necessary to the maintenance of the nutrition and functional activity of these organs. By virtue of their elasticity they effectually maintain the continuity of the actual energy expended by the ventricle, and also the uniformity of work performed by the organs which they supply. With the development of sclerosis the elasticity of the arterial wall is impaired, when the nutrition and functional activity of the organ supplied are apt to fail—a failure which is doubtless accentuated by diminished elasticity in the aorta. Hence it is that sclerosis of the arteries supplying the heart, brain (the circle of Willis) kidneys and liver, may lead to very important pathological developments—such as fatty and fibroid degeneration of the heart wall, vertigo and other symptoms referable to disturbance of the cerebral circulation, diminution of the urinary output (and possibly chronic fibroid degeneration of the kidney), hepatic inadequacy and fibrosis.

### *Use of the Term "Arterio-sclerosis."*

The term "arterio-sclerosis" is *now* generally employed to denominate any form of thickening of the arterial wall, whereas it was formerly restricted to sclerosis of the small arteries and arterioles. Now it embraces as well any thickening of the large arteries. From the point of view of pathological anatomy it is conceivable how this unification of all forms of arterial thickening should be preferred to the older dual classification of

* Mott, in 'Allbutt's System of Medicine,' vol. vi, p. 295, says: "I have pointed out that the openings of the fenestrated membrane probably serve the purpose of allowing nourishment to enter from this source (the vasa vasorum)."

* 'The Lancet,' vol. ii, 1906, p. 1334.

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atheroma and arterio-sclerosis; for the differentiating points between the two pathological states may be explained by the differences in structure of the large arteries and the arterioles—the former being supplied with vasa vasorum, and atheroma according to H. Martin is the result of arterio-sclerosis of those nutrient vessels.* But notwithstanding the want of clearly defined pathological differentiations between the various forms of thickening (atheroma, arterio-sclerosis, endarteritis), there is no doubt from the clinician's point of view that the recognition of these forms as separate entities is well supported by evidence furnished during life; and is, moreover, useful in enabling us to define certain clinical groups of arterial thickening.

*Different Forms of Arterial Thickening.*

Consequently I shall adhere to the dual division of arterial thickening—atheroma and arterio-sclerosis. At the same time the clinical study of these primary groups may furnish us with some other subsidiary divisions.

I agree with Clifford Allbutt,† who believes that there are several diseases of the arterial wall which we denominate by the current denominations; and Metchnikoff holds the same view: "Probably diseases of the arteries of different kinds, and arising from different causes, are grouped under the terms atheroma and arterio-sclerosis."‡ Pathologists have for years recognised several distinctive forms of arterial thickening, such as those due to obliterative arteritis, and the endarteritis of syphilis and of the infective fevers. But apart from these forms of endarteritis the cases of arterial thickening commonly met with in practice are due to what we call atheroma and arterio-sclerosis; and it is clinically useful to recognise these types of thickening as constituting two primary generic groups and forming, as they do, at any rate, a provisional basis for future observation and experience.

*Atheroma.*

Atheroma is, strictly speaking, an arterial disease of after life. The involutionary process which produces the thickening begins in the non-vascular portion of the arterial wall—the intima—from which it extends to the media. Other non-vascular structures—such as the cornea, the lens, cartilages—are apt to be the seat of senescent degenerative changes. Though atheromatous thickening is usually met with in the systemic arteries in advanced age, its appearance is by no means proportional to age. For example, I have met with several subjects of from seventy-five to eighty-five in whom the accessible arteries were quite free or practically free from it; and in one case I found it but slightly appreciable even in his hundredth year. On the other hand, you will have noted its pronounced existence at much earlier periods of life. In such cases when the thickening invades most of the accessible arteries it indicates, as a rule, presenility of the arterial system; but when it is smooth and uniform and is localised in the arteries of the forearm, and more especially in those of the more active side, such as the right side or the left in the left-handed, it may be only due to work which thickens the arterial wall. Therefore, you should not infer that every thickened radial implies atheroma; it merely suggests inquiry. In a large proportion of the cases of atheromatous thickening met with in clinical observation, even when most of the accessible arteries are involved, we fail to meet with any distinctive symptoms or indications of disordered health. In all such cases it would seem that the atheroma invades chiefly or only those arteries which serve as conduits of the blood to the limbs and the other somatic areas; and so long as it is thus limited the health may remain without appreciable impairment for very many years. On the other hand, atheroma may present itself as a disease of some gravity—threatening life and terminating fatally. Let me refer to some of the conditions in which atheroma becomes a formidable pathological factor.

(1) When atheroma seriously invades the arteries supplying organs of prime importance to life—such as the brain, the heart, the kidneys and the liver—we may get various clinical pictures of different forms of serious disease. There is reason to believe that in certain cases the visceral arteries may be affected much in advance of those supplying the limbs; and that the coronary and cerebral arteries may be exclusively invaded. Of the visceral arteries the coronaries are the most

* 'L'Artériosclérose et son Traitement,' par Prof. Gouget, Paris, 1907.

† 'Brit. Med. Journ.,' 1907, vol. i, p. 1392.

‡ 'The Prolongation of Life,' by Elie Metchnikoff, English translation by P. Chas. Mitchell, M.A., D.Sc., F.R.S., etc. The title of the original work is 'Étude sur la Vieillesse' (Essais Optimistes) Paris, 1907.

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frequently affected; then follow the renal and the cerebral arteries and the branches of the cœliac axis.*

(2) When atheroma is associated with a super-normal arterial pressure along with evidence of considerable cardiac strain in maintaining that pressure, the gravity of the case rises considerably.

(3) Another unfavourable state of the circulation in atheroma with a super-normal pressure is considerable distension of the arteries, as shown by a greatly increased calibre of the radial artery measured by the arteriometer. This dilatation is caused by the continued stress of the pressure overcoming the onus of the arterial wall; at the same time the pressure remains moderately high from the persistence of increased resistance ahead.

(4) Hypertonus is quite a frequent and menacing concomitant of atheroma; and it is apt to produce rapid increments of the arterial pressure which may result in hæmorrhage (*e. g.* cerebral) or over-strain and failure of the heart muscle with dilatation. When the heart gives out we have, as a rule, the precursory development of hypertrophy, which is the tell-tale of the prolonged struggle of the centric bundle of muscle-fibres (the ventricle) with the increased peripheral resistance maintained by the hypertonic state of the distal muscular fibres.

(5) The gravity of atheroma is greatly increased by the involvement of the aorta, the elasticity of the walls becoming impaired with dilatation of the vessel or the production of aneurysm. Dilatation of the aorta is a common result of atheroma *plus* hyper-normal arterial pressure. You should bear in mind that the aorta may be the seat of atheroma, and even of advanced atheroma, while the arteries of the limbs may be free from it; and also that the aorta stands in the front rank of the arteries most frequently affected.

(6) The fact of syphilis in the history of a case of atheroma as a rule adds to its gravity.

### *Atheroma Develops from Foci.*

Many pathologists regard the focal development of atheroma as a distinctive feature of this form of arterial thickening. It begins in small isolated points or patches which may ultimately coalesce; and as a rule the thickening is felt to be markedly irregular and nodular.

* Gouget, *op. cit.*

### *Atheroma is Less or More Local.*

Besides its focal development another distinguishing feature of atheroma is its local distribution. As a rule it begins in, and generally confines itself to, the central and medium-sized arteries, in which it is unevenly distributed. I have pointed out that the accessible arteries may not be thickened or may be thickened to a slight degree only, though the arteries not open to direct observation during life may be the seat of decided or advanced atheroma—such as the aorta, the coronary and cerebral arteries. Such cases, however, form the exception rather than the rule.

### *Calcareous Arteries.*

The deposition of lime in the walls of the arteries is perhaps more common in atheroma than in most other forms of arterial thickening. Some recent writers refer the calcification of the arteries, cartilages, etc., of the aged to transference of lime from the bones. Durand-Fardel and Sauvage emphasise the coincidence between atheromatous calcareous lesions and senile degeneration of the bones; and Metchnikoff shows that the transference is effected by the agency of polynucleated cells (osteoclasts) which develop in the bony matter of the aged, dissolve out the lime salts in the skeleton (probably by secreting some acid) and pass them over to the circulation, whence they are deposited in the walls of the arteries, etc.

"There is no disharmony," says Metchnikoff, "in the nature of old people so striking as the transference of the lime salts from the skeleton to the blood-vessels, producing as it does a dangerous softening of the former and a hardening of the latter that interferes with their function of carrying nutrition to the organs. It is the manifestation of an extraordinary disturbance of the properties of the cells that compose the body."*

In a recent paper,† W. Blair Bell regards calcification as a reparative process—the calcium salts being transferred by the agency of leucocytes—and certainly it is common in all forms of diseased or necrosed tissues. As somewhat opposed to the views of Metchnikoff we have the observations of Dhéré and Grimmé that in dogs the proportion of lime

* *Op. cit.*, p. 31.

† "The Physiological Importance of the Calcium Salts," 'Brit. Med. Journ.,' 1908, vol. i, p. 505.

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in the blood diminishes with age*; and the fact that calcification is not confined to the after years of life. The middle coat of the artery is the principal seat of the deposition of lime, where it must surely obstruct the normally feeble circulation of blood and perfusion of lymph to the intima through the fenestræ of the membrane of Henle. Hence calcification may greatly favour the necrosis and roughening of the intima and the production of thrombosis.

*Calcification not a Necessary Result of Senescence.*

Calcification of the arteries is not, however, an inevitable fact in old age or even in subjects who attain a great age. I have myself observed the absence of calcified arteries in many subjects whose age varied from 75 to 100. In one (Sir H. Pitman) who has now attained his 100th year, the accessible arteries are free from calcification and are almost free from sclerosis†; and Metchnikoff, referring to Madame Robineau in her 107th year, observes, "The most remarkable circumstance was the abscence of sclerosis in the arteries, although such degeneration is generally believed to be a normal character of old age."‡

Even in the post-mortem record of Thomas Parr, æt. 152 years and 9 months, by our illustrious Harvey, we find this interesting note: "In raising the sternum, the cartilages of the ribs were not found harder or converted into bone in any greater degree than they are in ordinary men; on the contrary, they were soft and flexible."§

We may therefore infer that if decalcification of the bones is a normal process in old people, it does not follow that the lime thence abstracted is necessarily deposited in the arterial walls or in the cartilages; it may be eliminated by the kidneys.

*Atheroma in Relation to Senile Vigour and Activity.*

According to my observation on the senile I am led to conclude that the absence of, or the sparse development of, atheroma in the aged is, as a rule, associated with a somewhat greater retention of bodily and mental activity than obtains when arterial thickening is appreciably developed. In such cases the brain is generally quicker to apprehend and to respond, the memory is less impaired, and the movements are more elastic and free than in those in whom the arteries are thickened. At the same time it frequently happens that thickening of the systemic arteries is nevertheless consistent with length of days and a vigorous and active old age, providing the arterial blood-pressure remains normal or is not much increased, and providing the thickening is limited to the arteries which supply the systemic muscles and other somatic tissues, etc., and does not involve those distributing the blood to the brain, heart, or the organs in the splanchnic area. Gouget pithily and truly says: "It is certain nevertheless that one dies more often with arterio-sclerosis than from arterio-sclerosis," (the term "arterio-sclerosis" being employed so as to include "atheroma").*

*The Combination of Atheroma with Arterio-sclerosis.*

Atheroma thus limited to the somatic area (limbs, etc.) generally develops slowly and in a latent manner, there being no distinctive symptoms beyond, perhaps, slight indications of deterioration of physique in some cases. Such symptomless cases form a marked contrast to those in which we have the actual clinical picture of diffuse arterio-sclerosis. In this important group of cases we have unquestionable indications of an abnormal state of the circulation, and to account for it we may suppose that one or other of the following pathological conditions supervenes: we may have a widespread development of atheroma extending from the centric to the peripheral vessels (systemic and splanchnic); or we may have the involvement of the distal vessels by diffuse arterio-sclerosis, which complicates the more limited atheromatous thickening in the larger arteries—the two pathological processes (if they be different) being consecutive to each other or developing simultaneously. Whichever be the explanation—and the correct interpretation is of little practical importance—the cases are indistinguishable in symptoms and course from those in which arterio-sclerosis begins as a generalised peripheral disease, and may in fact be regarded as examples of this disease notwithstanding the presence or pre-existence of atheroma.

* Quoted by Gouget, *op. cit.*

† 'Studies in Blood-pressure,' 2nd edition, 1908.

‡ *Op. cit.*, p. 7.

§ "The Circulation of the Blood," with an Introduction, by E. A. Parkyn, p. 208.

* *Op. cit.*

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Clinical observation has led me to think that in many such cases senile thickening of the arteries is due not merely to atheroma pure and simple, but to a combination of it with diffused arterio-sclerosis, which is the predominant clinical or pathological factor.

### *Arterio-sclerosis generally Develops Earlier than Atheroma.*

Arterio-sclerosis may be said broadly to belong rather to middle life than to the period of senescence. Certainly many typical examples of it begin and develop to an advanced stage before the declining years of life set in. In fact much has been written on the appearance of arterio-sclerosis quite early in life.* Undoubted, though comparatively rare cases of sclerosed and calcified arteries have been met with, not only during adolescence, but even in infancy; and cases of congenital arterio-sclerosis have been observed. But in a large proportion of such cases the course is infective—syphilis or the infective fevers.

### *The Thickening of Arterio-sclerosis is generally Less Uneven than that of Atheroma.*

Clinically, arterio-sclerosis has two rather distinctive features—namely, its mode of development and its distribution. It develops, as a rule, at first less unevenly than atheroma and it selects large districts or even the whole of the arterial system. It cannot, however, be said that the thickening it produces is *always* less irregular than that of atheroma; for we often meet with much nodulation and corrugation, as in endarteritis obliterans and in the forms of arterio-sclerosis produced by lead—especially when the arteries have become calcified. Then again, in atheroma the thickening in individual arteries may resemble in uniformity or in diminished irregularity the thickening of arterio-sclerosis.

### *Arterio-sclerosis is Primarily a Diffuse Peripheral Disease.*

Clinical observation seems to me to point rather to the early or even primary invasion of the peripheral part of the circulation by arterio-sclerosis and the subsequent extension of it towards the central part of the arterial system; whereas in atheroma we find that the central and medium-sized arteries are more frequently affected in the first instance. But the demonstration of the early peripheral development of arterio-sclerosis is by no means an easy matter; for in nearly every case which comes under observation for the first time arterial thickening of the accessible arteries has already taken place, and it is only in the exceptional case are we afforded opportunities for consecutive observations of the early stages of the disease. I think it is not improbable that in many cases the disease actually begins as a generalised disease—invading the whole arterial system at the same time. But this view is quite consistent with the distal origin of arterio-sclerosis; for the arterial wall itself is just as much a part of the peripheral area of the circulation as any other structure of the body. Notwithstanding the wide diffusion and symmetrical development of the disease there is no doubt that there is considerable disparity in the amount of thickening in the arteries of different areas, such as the arms and forearms, etc.

I think that most pathologists are of opinion that the disease begins in the media and may extend to the intima and adventitia.*

### *May Arterial Thickening be regarded as a Defensive Lesion?*

It may be well to keep in mind that arterial thickening may serve the purpose of a protective lesion by resisting the expansion of the arterial wall under the stress of increased blood-pressure—or a pressure in excess of the resistance, which the normal wall can summon to prevent distension of the calibre. The resistance of the arterial wall may be strengthened in three ways; either by hypertrophy of the musculature—as shown by the important work of Savill† on arterial hypermyotrophy—or by proliferation of the fibrous tissue, or by deposition of lime salts. In some cases calcification may serve as the final defensive effort of nature to limit expansion of the calibre.

(*To be concluded.*)

*September 7th*, 1908.

* See Dr. F. Fremont Smith in 'American Journal of the Medical Sciences,' February, 1908.

* See Savill, 'Path. Soc. Trans. London,' 1904; also Dixon, 'Proceedings Royal Society of Medicine,' 1908.

† 'Trans. Path. Soc. of London,' 1904.

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# CONGENITAL PYLORIC STENOSIS.

A Lecture delivered at the Hospital for Sick Children, Great Ormond Street, W.C.

By ROBERT HUTCHISON, M.D., F.R.C.P.

CONGENITAL hypertrophy of the pylorus, or congenital hypertrophic stenosis as it is sometimes termed, the subject of my lecture this afternoon, is a condition which has only been generally recognised in the last decade or so, but which is probably very much commoner than is generally believed, and I am perfectly sure that anyone who is capable of diagnosing it will meet with cases in his practice not very infrequently. Inasmuch as the treatment of this condition, if it is to be successful, must be begun early, it is extremely important that you should be able to diagnose it when you see it, and that is why I have taken this subject for my lecture to-day.

## SYMPTOMS.

I shall not trouble you with the history of our knowledge of congenital pyloric stenosis, because that really does not throw any particular light upon the nature of the condition, and it is of no use to you in your practice, so I shall begin at once with the symptoms, which, fortunately, are few and definite. The kind of case which exhibits congenital pyloric stenosis is this. The child is usually—indeed I may say nine times out of ten—a boy, and he is brought to you when a few weeks old because, as the mother says, she cannot find any food to suit him at all. She will complain that, no matter what food she tries, it results in the production of vomiting. You will usually find that a great many different foods have been tried—many of the patients, indeed, have run the whole gamut of the patent infant foods in the market—and yet the vomiting goes on. There is a very curious fact about the vomiting, and it is this, that when a new food is tried it will often seem to suit the child for a period of about twenty-four hours or a little longer, and then the vomiting begins again, and will go on every day until a new food is tried, when it will again often cease for a day or two only to re-commence once more. At each of these occurrences the hopes of the mother are buoyed up; she believes she has at last found the kind of food that is going to suit; but they are immediately dashed to the ground again when the vomiting re-asserts itself. Why it is that the new food is often borne for a time in these cases I cannot tell you; it is one of the many curious clinical facts that characterise this condition.

On careful inquiry you will usually find that the vomiting has not strictly dated from the moment of birth. As a rule you will be told that the child was born a fine, healthy child; that he was fed from the breast at first, and all went well for a week, ten days, or a fortnight, but then vomiting began, and the child was taken off the breast, very often by medical advice, because of the continuous sickness, and put on to some artificial food. As that was also followed by vomiting, another artificial food was tried; and so on.

I want you to observe—because it has some bearing upon the view that one takes of the pathology of the condition—that the vomiting is not, strictly speaking, congenital; it is more usual for it to begin ten days or a fortnight after birth. In addition, the vomiting has certain peculiarities which characterise it and distinguish it from the ordinary vomiting of dyspepsia. In the first place it is often extremely violent, or what is termed "projectile"; that is to say, the child will suddenly throw out the stomach contents forcibly, often projecting them quite a long way from itself. This forcible or projectile character is one of the things that distinguishes the vomiting of congenital pyloric stenosis from that which is due to ordinary dyspepsia. The vomiting is also characterised by its abundance, and the mother will tell you that the child brings up much more food than it swallowed at its last feed. Further, you will often be told that the child brings up a quantity of "slime" with the vomit. Slime is what you and I know as mucus, and the bringing up of a quantity of mucus is another of the things which characterises this particular kind of vomiting.

Another prominent symptom that the child will exhibit besides the characteristic vomiting is *progressive loss of weight*. That, of course, is natural. If the child is constantly vomiting it naturally loses weight, and along with the loss of weight there is a shrivelling up of the skin, and a loss of its elasticity, such as you meet with in cases of chronic diarrhœa in children. The loss of weight may proceed until it reaches a very serious

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degree; such children may waste away until they are literally mere skin and bone.

The third thing which characterises a case of pyloric stenosis is that the child is usually, indeed almost invariably, *constipated*. That is a sign of very great importance, because if a child is vomiting from unsuitability of food, instead of being constipated it usually has diarrhœa; and a combination of vomiting *plus* constipation should always arouse in your minds strong suspicions that you are dealing with a case of this sort. The reason for the constipation in this case is obvious. If no food, or very little food, is entering the intestines, obviously there must be very little in the way of excreta to go out of the intestines, and the constipation, therefore, is what is sometimes called the "constipation of starvation." It is the inevitable result of the small amount of nutriment which is finding its way through the pylorus into the bowel.

### Physical Signs.

When you come to examine the child for physical signs you will be at once struck by the *emaciation*. Observe that, although these children are emaciated, they are not cachectic, and that is a point worth noting. They show the thinness of starvation but not the thinness of cachexia; in other words, although thin, often miserably so, they are not anæmic, and you know that it is a combination of emaciation with anæmia which makes up cachexia.

In examining the abdomen, you will usually find that there is a certain amount of fulness in the upper part above the umbilicus, whereas the rest of the abdomen is sunken. And then, having your suspicions aroused, you should look for what is, after all, the most characteristic sign of the condition, viz. *visible stomach peristalsis*. If you are going to succeed in seeing visible stomach peristalsis you must look for it when the stomach is full. There is no use in examining a child for gastric peristalsis shortly after it has been sick; if you wish to make it out you should always insist upon the child being fed in your presence, and immediately it has taken its food you should make your examination. If the peristalsis be not immediately visible you may sometimes elicit it by applying friction over the stomach; massaging the stomach may bring it out. This visible peristalsis assumes a very characteristic form. You will see appear below the left costal margin a swelling about the size of a golf ball, which will begin to travel downwards to the right, sweeping above or even over the umbilicus, and which, before it has disappeared, is followed by another swelling, so that a dumbbell or hour-glass-like appearance is produced over the upper part of the abdomen. Sometimes in extreme cases you will see three such swellings, or in other words, three waves of peristalsis, simultaneously visible over the stomach. Such peristalsis, *if it be well marked*, is absolutely pathognomonic of the condition, and sometimes it is extremely well marked. I have seen it so well marked that the swelling of the stomach could be seen through the child's clothing. Unless it be well marked you should not attach importance to it, because *slight* gastric peristalsis may be visible as a purely transitory phenomenon in circumstances other than those in which there is an actual constriction of the pylorus.

In addition to the emaciation and the visible peristalsis you will find in many, but by no means in all, of these cases that you can actually feel the *thickened pylorus*; and you can feel it usually to the right of the umbilicus. Observe that sometimes the pylorus cannot be felt, even in undoubted cases, for the simple reason that it lies tucked up under the liver where you cannot get at it. But if you wish to give yourself every chance in feeling for it there is a particular way of setting about it, and that is that you should not merely press downwards and backwards, as one does in ordinary palpation of the abdomen, but you should pinch up the deep structures, and try and grasp the thickened pylorus between your fingers and thumb. You then find an elongated swelling about the size of a hazel nut, or a little larger; and sometimes if you are lucky you will feel it actually undergoing contractions whilst you feel it; you feel it undergoing relaxation, followed by hardening.

If you proceed further in your examination by washing out the stomach, you will usually be able to make out that the capacity of the stomach is increased, that instead of holding, let us say, a couple of ounces or so, which is normal for that time of life, the stomach holds three, four, or even more ounces of fluid. In other words, you can prove the existence of actual *gastric dilatation*, and that, in addition to the peristalsis and the palpable

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tumour, is the third purely stomach sign of this condition. So much, then, for the symptoms and signs.

### Pathology.

It will be well for us now to look for a moment at what is known as the pathology of congenital pyloric stenosis, and I may say at once as regards this subject that we do not know what the real pathology of the condition is. What we do know is that in those cases which prove fatal, or in which operation has been performed and an opportunity has been given of inspecting the pylorus during life, there is present a great thickening of the pylorus, which section shows to be due almost exclusively to hypertrophy of the circular muscular sphincter of the pylorus. I have here a few specimens which show this thickening very well. Here, for example, is one in which you can see quite well with the naked eye the ring of thick muscular tissue surrounding the pylorus, and which practically converts it into a tube of almost cartilaginous consistence. That is one thing you can make out. Another thing that you can find is that the whole wall of the stomach is more or less hypertrophied owing to the efforts of the stomach to drive its contents through this narrow channel. Thirdly, you will find that the mucous membrane of the stomach is in a state of chronic catarrh. That summarises all there is to be said about the morbid anatomy of the condition —there is an hypertrophy or overgrowth of the circular fibres of the pylorus, there is a thickening of the whole wall of the stomach, and there is catarrh of the mucous membrane.

So far everyone is agreed, but when you come to attempts to explain how these anatomical findings are produced you at once enter the sphere of very acute controversy. You may say that, roughly speaking, there are two views prevalent as to how it is that this condition is brought about. There is, first, the view that *the hypertrophy of the muscular fibres of the pylorus is a congenital anomaly*—that there is a true overgrowth, a true hypertrophy of the circular muscular fibres existing as a congenital anomaly, just as you may have a congenital club-foot or any other inborn malformation. The other chief view of the pathology of the condition is that the hypertrophy is not a congenital malformation at all, but is *consecutive to a spasm of the pylorus.* Those who adopt this view maintain that the condition of which we are speaking is, in its essence, a spasm of the pylorus of great severity and of long continuance, and that, as a consequence of the continued spasm, you get what you obtain from over-use of muscle anywhere—hypertrophy—and that that thick pylorus which I showed you in the specimen is simply the result of over-use—it is the result of prolonged and repeated spasm of the sphincter of the pylorus. When those who hold this view are asked how this spasm is brought about they are compelled to have recourse to various hypothetical explanations. Some assume that there is an "inco-ordination." Now, ladies and gentlemen, inco-ordination is one of those blessed words like "Mesopotamia," and "toxin," and "reflex-action," and a few others which are usually used as a cover for much ignorance; and it is no more satisfactory in this case that it usually is. Then there are some who say the spasm of the pylorus is brought about by an excess of acid in the stomach contents, because it is known that excess of acid in the stomach leads to the closing of the pylorus. No chemical proof has been advanced that such excessive acidity actually exists. Others say that something has gone wrong with the chemical mechanism in the duodenum, which is really responsible, as Pawlow has shown, for the normal opening and shutting of the pylorus. But all these explanations, I confess, are extremely unsatisfactory. My own view is that the condition is not a congenital anomaly, but that the second view is right, explain it how you may, and that we have here primarily to do with a spasm. In favour of that I would only point to the two great clinical facts which cannot be got over: first, that the symptoms do not come on immediately after birth, but generally a fortnight or more later, and second, that the condition is capable of being absolutely cured by purely medical treatment, leaving the stomach, as I have proved to my own satisfaction time and again, in a perfectly normal condition afterwards. I cannot see, on the assumption of a congenital anomaly of formation, how such cases should make a perfect recovery without operation and without any dilatation of the stomach being left. I believe, therefore, that we have here to do with a spasm which goes on to hypertrophy, but how that spasm is brought about I confess I do not know, and from the point of view of treatment I do not think it matters.

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## Treatment.

That brings me to speak of treatment. Now the first essential here is that you should begin treatment early. I believe that failure in these cases is due to the condition being recognised too late, with the result that treatment is left until the child is in such an exhausted condition that recovery is difficult, if not impossible. That just emphasises what I have already said about the importance of early diagnosis.

The first and most important of all things in treatment—I put it before diet and everything else—is systematic *washing out of the stomach.* That is *the* main factor in getting these cases well. You wash out in the ordinary way every day, or twice a day if need be. I do not think it much matters what you use to wash out the stomach with, but at first there is an advantage in using a weak alkaline solution—a weak solution of bicarbonate of soda, for example—because there is often so much mucus in the stomach owing to the prolonged catarrh of the mucous membrane, which has resulted from stagnation of the contents, that it is an advantage to use an alkaline wash to bring the mucus into solution and so get rid of it. I prefer, therefore, at the outset to use a drachm of bicarbonate of soda to a pint of warm water, and use it once or twice a day. The immediate result of the washing is that the vomiting ceases, or almost ceases; it will cease almost as soon as you have got rid of the mucus.

The second question that faces you in treatment is, *how you are to feed the child?* Your object must be to give food which will pass through the pylorus as easily as possible, and therefore you will select a milk which does not form large clots—that is obvious. As a matter of fact there is nothing better, when you can get it, than human milk, and that is why it is always a pity when these children have been weaned early, as they usually are, under the impression that breast milk is disagreeing with them. That is always a pity, because you will find when you wean them that you are jumping out of the frying-pan into the fire, for if these children are not able to retain human milk, the chances are they will not retain anything else. So that if the child is at the breast you should keep it at the breast; if it is off the breast it is a good plan to get a wet nurse. The practical difficulty of breast feeding is that these children need to be fed frequently and in small quantities, and that becomes a nuisance to the mother. In some cases you may have to draw off the breast milk by a pump, and feed the child with it from a bottle or by teaspoon at short intervals of, say, every hour. Assuming that human milk is unobtainable, I believe the next best thing is fully peptonised milk, given diluted with an equal quantity of water, and I do not believe that, beyond that, there is any good in searching about for a particular food that will suit these children. If you adopt lavage, and do it properly, you will find that they will retain their food well enough, but you will never find anything which will pass quite satisfactorily through the pylorus, at first at least. Hence, although there are many combinations on which these children will do well, such as whey and cream, whey and egg white, desiccated milk, malted milk, and so on, yet my experience has been that if they do not do well on peptonised milk they will not do better on anything else, and there is no use in perplexing yourself by a large choice of methods of feeding.

As regards the intervals of feeding, the frequency and the quantity, you have to be guided by the capacity of the stomach in the particular case with which you are dealing; you have to feel your way and to notice how much the child can stand without being sick. Sometimes you have to feed every half-hour, or every hour or two hours. Here, again, you will find that, once you start washing out, you will soon be able to get the child down to approximately the ordinary intervals which are natural and right for a healthy child of that age.

Third, as regards the question of *drugs*, I believe there is none which is of any real use in this condition. Opium in minute doses has been highly recommended, $\frac{1}{80}$th to $\frac{1}{40}$th of a minim of the tincture given shortly before each feed. These quantities seem almost infinitesimal, almost homœopathic, and yet I have known one of these children rendered almost comatose by $\frac{1}{20}$th of a minim of the tincture of opium given before each feed for less than a day. Owing to their exhausted and feeble condition they are extraordinarily sensitive to opium, but some, particularly German writers, have reported very great benefit from giving it, the theory being that it helps to abolish the spasm of the pylorus. Although I see no objection to trying

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opium, personally I have been disappointed in the results it has yielded. I have tried alkalies in a few cases, and I have tried belladonna, which has a great reputation as a relaxative, and in neither case have I found any benefit from so doing. The treatment, therefore, resolves itself into careful feeding and nursing, and persistent lavage of the stomach. You will be told sometimes that it is an advantage in these cases to feed *per rectum*, or rather to give water or saline solution *per rectum* in order to make up the deficiency of fluid in the body which undoubtedly exists. As to that I would only say that my experience has been that it is almost impossible to get children of that age to retain saline enemata for any length of time, and, therefore, although such treatment is perfectly right in theory, in practice it is almost impossible to carry it out.

### Prognosis and Course.

You may say, what are the results of such treatment? I can only tell you that, taking my own experience of these cases in private practice and confining myself to well-marked cases, about the diagnosis of which there could be no reasonable doubt—cases in which the gastric peristalsis was *very* well marked, in which there was great emaciation, and in many of which, at least, though I admit not in all, a thickened pylorus could be felt—I have had fourteen which were treated medically, and only one of those died, and curiously enough that was the only female child I have seen with this condition in private practice, in whom the condition began very early, and who died quite suddenly when less than three weeks old, a case in which we had not the opportunity of making a post-mortem, and as to which I have doubts as to whether it was an ordinary case of congenital pyloric stenosis. Even including that case, taking one's private cases only, the mortality has been one in fourteen; all the others have got well. And when I say well, I mean perfectly well. One has followed these cases out, some of them for three or four years, and they have remained perfectly healthy children. The question is, are they left with any dilatation of the stomach at all? I would say that, in the majority of cases, so far as I have seen they are left with no dilatation of the stomach; the condition seems to be one of genuine cure. But every now and then you will meet people in adult life who have stomach symptoms, signs of dilated stomach particularly, who date their symptoms right back to childhood, and who are, I believe, cases of imperfectly recovered congenital pyloric hypertrophy. I saw last week a young lady whose gastric symptoms went right back to childhood. Her mother told me that when she was two months old she was reduced to a skeleton, and was constantly sick; they could not get a food to suit her. She ultimately recovered, but she has suffered more or less from dyspepsia ever since, and at the age of five it was noticed that her stomach was dilated. I believe that was a case where one had to do with congenital hypertrophy of the pylorus, which had not completely disappeared. But that is an exceptional occurrence; the great majority of those I have seen do get perfectly well. I do not profess to explain how it is that such treatment cures those cases; I only say the fact that they are cured is, to my mind, an argument that it is a condition of spasm and not of true hypertrophy, and that if you can get rid of the spasm the muscle will go back to its normal size.

There are certain things you must be prepared for in the course of your treatment. I told you that almost as soon as you begin lavage the vomiting will cease, but you must not expect that as soon as the vomiting ceases the child will start to gain weight. Quite the contrary is the case. You should warn the parents that these children will often, after they cease vomiting, go on losing weight, and even to a large amount, for a considerable time. What happens, in my experience, is something like this: The vomiting stops and yet the weight continues to go down, and down, and down; everybody gets alarmed and begins to talk wildly about the necessity for operation. You must keep calm and say it is all right, that it is the natural course of things. The child goes on losing weight until it is four and a half or five months old, and then, as Browning says, "sudden the worst turns the best to the brave, the black minute's at end," it is round the corner almost at once, and the weight begins to go up. A very experienced sister in this hospital, who has seen a lot of these cases, once told me that she preferred those children who went rapidly down hill. On one occasion she advocated the child being sent out in order that it might come back much worse,

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"because," she said· "when they get to a certain pitch of badness then they generally get round the corner." It almost seems that when they get to a sufficient degree of weakness the spasm passes off, and it is because people are not aware of this natural course of the disease, even without treatment, that operation is advocated. You may ask, What is your view as to operation? My view is that *operation is never in any circumstances justified in these cases.* I have told you that my own mortality in cases treated in private by medical methods has been one out of fourteen, and the operative mortality in the hands of the best and most experienced surgeons is admittedly 50 per cent. So long as one can cure thirteen out of fourteen by medical measures, and so long as the best surgeons go on losing 50 per cent. by operation, you can see for yourselves that operation is not a thing to be recommended. It is not to be recommended for this reason, that you never know, and in the nature of things never can know, when you would be justified in recommending it. There is no point at which one of these children is so bad that you can say it is impossible the child should recover under medical treatment. I have known these children hanging between life and death, on the very brink of the grave, for weeks, and finally get perfectly well, and if you wait until such time as you can say, This child is certainly not going to get well under medical treatment; in other words, wait till the child is moribund, then, of course, operation is out of the question. So that you see you are placed in a dilemma. Although what I say is disputed by some people at present, I am perfectly certain that a few years hence there will be unanimity that in these cases there is no necessity to have recourse to any surgical operation whatever. Therefore I do not consider with you the relative advantages of such procedures as pyloroplasty, or pylorectomy, or gastro-enterostomy, because my deliberate opinion is that there is no necessity to have recourse to them in any case.

I have tried to put before you the symptoms, the pathology and the treatment of this condition, and my last word to you would be again that, if you are to be successful in treating these cases you must recognise them early. I freely admit that my own hospital statistics do not show anything like as good results as my private statistics, and the simple reason is that you do not get them early enough in hospital. When they do turn up it is very difficult to get them into the wards, and you have to treat them as out-patients; lavage is imperfectly carried out, the child is not properly nursed and fed, and it gets rapidly and badly into an exhausted condition, and very often when it gets into that condition chronic diarrhœa supervenes, and if one of these children gets chronic enteritis I believe recovery is rarely possible.

Above all I recommend you to keep your heads in the treatment of those cases. There is no class of case in which you need a greater share of equanimity. You will be naturally urged by the mother, who sees the child's weight dropping week by week, to do something more, to try another food, to give another medicine, to consider operation, to do something desperate to stop this state of things. It is your business to say: "No, this is the natural course of things, and I am perfectly prepared for it. It is what I would expect, and if you will have patience you will find the child will get round the corner, and everything will be well." I have had to take that attitude many times, and almost to hold people back from rushing at these children with the knife, and in every case I have been justified by the event, and I have no doubt at all that you will be so justified too.

*September 7th*, 1908.

**Acute Peritonitis.**—In the course of a report on the experiences with acute peritonitis at the Moabit Hospital at Berlin, Federmann states that diffuse gonorrhœal peritonitis is liable to heal spontaneously, as he has observed many times. It consequently does not require an early operation like peritonitis of other origin. Differentiation is often difficult, as the onset is stormy and the symptoms are grave. Exploratory puncture through the posterior vaginal vault will reveal the presence of gonococci in a rapidly made, dry preparation. In his experience the patients with acute peritonitis and a leucocytosis of 20,000 and over, operated on in the intermediate stage, all recovered. Leucocytosis of 15,000 or under by the third day, he says, contra-indicates operative treatment for the time being, as the resisting powers are exhausted. He has never witnessed recovery in any case of suppurative peritonitis with low leucocytosis after the expiration of the early phase.—*Journ. A.M.A.*, vol. li, No. 7.

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# SOME CLINICAL ASPECTS OF THE CHOICE OF ANÆSTHETICS, WITH SPECIAL REFERENCE TO THE C.E. MIXTURE.*

By L. ERASMUS ELLIS, M.D.

MR. PRESIDENT AND GENTLEMEN,—The subject of anæsthetics to which I have ventured to call your attention for a short time does not I think, need much apology, for it is one which either directly or indirectly affects every medical man and so will probably be of interest to us all.

This branch of medicine is, to the uninitiated, likely to prove very deceptive, for one is liable to feel considerable confidence at the commencement of one's experiences, but this assurance is likely to be greatly modified upon a closer acquaintance, until one finally recognises that the whole subject is beset with difficulties and is never thoroughly learnt, and I think it is not too much to say that one may glean some fresh point, some little variation of application or result, from almost every anæsthetic that one administers, and this would I believe still be the case did one continue to follow this branch of medical science for the whole term of a lengthy natural life.

Anæsthetics have, during the last decade, in common with other branches of medicine, made considerable advances, both in the variation of the agent used and in the apparatus for administering it. A short time may, I think, be profitably spent in considering some of the different varieties of anæsthetics and the appropriate choice to make in the various conditions one may be called upon to meet.

Anæsthetics roughly arrange themselves into two main classes :

(1) Those required for short operations; and—

(2) Those required for more lengthy ones, in which also a deeper anæsthesia is usually necessary, and I would ask you to kindly bear with me while I attempt a few remarks on the choice of anæsthetics in more or less major operations.

In the earlier days of his experience every man is liable to have a bias in favour of the particular drug and its special method of application, that he has met with mostly during his academical training, but every one should, I think, sink this bias as much as possible and approach each case upon its own merits In doing so one naturally thinks of the two main drugs used, viz. ether and chloroform, and around these may be grouped the different mixtures and sequences which have recently so much come into vogue. One does not nowadays begin with a certain drug, and irrespective of the patient's condition continue to administer that drug to the bitter end, but one as it were feels one's way, and gives the patient the particular drug which is indicated by his condition, and should the latter vary considerably so should the agent administered also vary.

Speaking generally, however, one should use as a routine anæsthetic the drug which is safest for the patient, and that drug is undoubtedly ether. Chloroform, the anæsthetic which usually forms the alternative with ether, is much more lethal in its results. I do not propose to weary you with statistics, but these latter definitely indicate that ether is the safest anæsthetic we have for prolonged administration and that it is from five to seven times more safe than chloroform.

Some objections to ether are, however, raised. Thus extremes of temperature cause it to be either too volatile or not sufficiently so. In the latter case the ether chamber (should a Clover's apparatus be in use) may be kept warm artificially, though practically this objection need not be regarded, since where an operation takes place, there a temperature sufficiently high to volatilise the ether usually obtains. On the other hand it is sometimes a little difficult to maintain anæsthesia with the ether boiling in the apparatus, which it does at 95° F., and so conceivably this may occur in hot weather, yet in most cases it is possible to keep an ordinary patient sufficiently under the influence of the drug.

A very substantial reason for excluding ether, however, is where a naked light or a thermo-cautery is to be employed, while the physical condition or the age of the patient likewise occasionally debars ether. Thus young children are sometimes better treated with some other anæsthetic, while the aged should not receive ether, nor those suffering from pleural, bronchial or pulmonary disease, notably phthisis. In regard to pulmonary tuberculosis, however, opinions seem to differ, for I saw a letter

* Being remarks delivered before the British Medical Association, Leicestershire and Rutland Branch.

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in a recent issue of a standard medical periodical* advocating the trial of ether application to the extent of complete anæsthesia as a *treatment* for phthisis. We have, however, high authority in support of discarding ether in this disease. These cases will often have to be treated with chloroform, but an extremely useful anæsthetic here is a mixture of two parts (by volume) of chloroform, with three parts of ether, the so-called C.E. mixture, to which I propose to revert presently. Some cases, however, which will not admit of a whole anæsthetic of ether, will sometimes allow induction with this drug and a continuation with chloroform. Should even this not be permissible and induction has to be made with chloroform, I usually like to have some ether in a semi-open apparatus such as a Rendle's mask, and apply this should any struggling occur, returning to chloroform as soon as the excitement stage is over. Most patients will admit of this small amount of ether and it frequently tides the heart over the most dangerous phase of the anæsthetic, and has a markedly quieting effect upon the patient.

The occurrence of ether tremor sometimes requires a change to be made, and this complication has the awkward habit of taking place in such operations as removal of varicose veins from the lower extremities, when the absence of movement is essential.

Stout, plethoric, or cyanotic individuals prove, as a rule, bad subjects for ether. If the administration of this drug be attempted with such patients it should be given with a large quantity of air. As illustrating the fallacy of laying down rules for the administration of anæsthetics, however, I may quote the case of a stout plethoric man of about forty whom I was recently called upon to anæsthetise and who gave no trouble when treated with ether in an ordinary Clover's inhaler, but took the anæsthetic perfectly and preserved a good colour with ease. As he was suffering from diabetes I felt debarred from giving him chloroform or any of its mixtures, as being likely to produce post-anæsthetic diabetic coma.

Cases of obstructed respiration such as occur with œdema of the glottis, pressure on the trachea, etc., should never be treated with ether, nor with any closed apparatus.

I have thus apparently excluded ether from a large number of cases, but I still maintain that it is in general the best and safest anæsthetic one can adopt.

For purposes of fine adjustment chlorolorm has the advantage over ether, and here the ether-chloroform sequence is useful. In many cases also the ether-C.E. sequence is of considerable benefit. In all these sequences, however, some experience is advisable, for small points of great importance occur which are not met with in the administration of a single drug. To illustrate my meaning, imagine that one is changing from ether to chloroform; it is advisable, provided other circumstances will admit of it, to allow the patient to partially come round from ether before applying chloroform; in fact I prefer the patient to cough and thus clear any collection of mucus that may have occurred in the trachea, because this slight obstruction by mucus may be unsafe with chloroform, though it may be disregarded when using ether, and likewise the patient may inhale too much chloroform while the ether respiratory stimulation still exists.

I mentioned the C.E. mixture as being a useful anæsthetic. This has in some institutions largely taken the place of the A.C.E. mixture which has been in vogue for some time, and I think wisely so, for the alcohol fulfilled no special office, the mixture being as stable without it as with it, while some authorities hold that the alcohol increases the after-effects.

C.E. takes an intermediate place between ether and chloroform as well in toxicity as in action, and many patients who cannot take either of the last-mentioned drugs may safely be treated with C.E. Children take this mixture well, and it is much safer for them than chloroform, which is erroneously considered by some to be less toxic with children than with adults. This fallacy arises owing to complications being less frequent with children because of their weaker muscular development; they also recover from states of depression of both respiration and circulation that in adults would prove more difficult to cope with, but they do succumb sometimes to this drug, as proved by certain statistics quoted by Comte, wherein he mentions twenty-one deaths from chloroform occurring in children out of a total of 232 fatal cases collected by him Weir, on the other hand,

* 'Lancet,' September 14th, 1907.

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stated some years ago that no deaths under ether had occurred up to that time in children under twelve years of age, but then again, ether is sometimes objected to because the upper air-passages in these small people are so sensitive that the breath is held to a troublesome extent during the administration of this drug.

Another class of patients for whom the C.E. mixture is eminently suited is that of advanced cases of morbus cordis, for whom the closed ether apparatus is bad, while the toxic effects of pure chloroform are to be feared. Cases of fatty degeneration, atheroma and pericarditis do well with this anæsthetic. In cerebral cases also it is most useful. I had cause somewhat recently to subject C.E. to a severe test in the case of a cerebral operation which lasted some two hours. I had no reason to change from this anæsthetic and it proved very satisfactory. The patient was certainly a good deal collapsed for some time afterwards, but the operation was lengthy and of a very grave nature. For cases of exhaustion, shock or collapse C.E. may be recommended, but while the heart's action may at the time be improved by this anæsthetic it is likely to become worse afterwards, and provision should be made accordingly.

A good proof of the suitability of C.E. was given in the case of a child of seven I recently treated with this mixture. She had been very ill for several days with cerebral symptoms and mastoidectomy was ultimately decided upon. I must admit I approached this case with considerable misgiving, for the child was in an extremely grave and asthenic condition. She, however, took the C.E. mixture easily, gave no anxiety from the anæsthetist's point of view all through the operation, which lasted upwards of an hour, and was not badly collapsed afterwards.

The mixture of chloroform and ether is said to be the best anæsthetic to use during the course of pregnancy, while it is very good for the operation for the removal of tonsils and adenoids in children, especially those under four to five years of age; whereas in the operation for amputation of the breast, when the stage is reached just prior to the actual removal of the organ, when shock is so liable to take place, C.E. proves most serviceable, while obese patients and the aged take this anæsthetic well.

Its method of administration is not difficult. It may be applied on an ordinary Skinner's mask to children, the aged and infirm, and those asthenic cases suffering from advanced disease which do not require much anæsthetic, and also where only a light degree of unconsciousness is required. One also finds that some women and certain subjects who are easily anæsthetised take it adequately in this open method, but for healthy, vigorous individuals, a partially-closed apparatus may be necessary. This may be met by a felt cone with a sponge in the apex, or a Rendle's mask, but a still better apparatus is one invented by Dr. Frederick Hewitt some two years ago (see illustration), consisting of a face-piece similar to the one which belongs to his wide-bore modification of Clover's ether inhaler, attached to a short, wide metal cylinder placed at right angles to the mouth-piece. containing a loose-meshed sponge and having a diaphragm closing its lower end and a wide opening at the top, through which the C.E. mixture is dropped or poured on to the sponge. With this apparatus the entry of air is so free that it is difficult to give an overdose of this anæsthetic, while should a more concentrated vapour be required it may be obtained by simply placing the hand lightly over the opening of the cylinder and thus reducing the entry of air.

Dr. Hewitt's C.E. inhaler.

It will be found that patients who are weak and wasted by disease take this mixture better than healthy, strong, and vigorous individuals, but this holds with every anæsthetic, and I always maintain that this latter class of person, represented for instance by the strong, muscular, well-developed man, is far more difficult to deal with than the weak and debilitated patient, or even the one suffering from grave organic disease.

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It may be gathered from what I have said that the C.E. mixture serves well for almost every kind of grave and advanced disease, and for those cases where the greatest risk is run in giving an anæsthetic, and it is mainly due to its being used largely in these serious and difficult cases that statistically it may not appear in as favourable a light as it ought to. There is, however, I think, little doubt that it is one of the safest and most useful anæsthetics we possess. It is not unpleasant to the patient, and its administration is fairly simple, while the apparatus necessary is not of a cumbersome nature. It therefore, from many points of view, adapts itself particularly well to the requirements of country districts and to the uses of private practice in general.

*September 7th*, 1908.

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Mr. Chr. Barthel, in a paper on the enzymic reactions of cow's milk and their importance for the hygienic control of milk, drew attention to the test described by the Dane, Professor Storch, by which it is possible to detect whether a milk sample has been heated to 80° C. (pasteurised) or not, as one of the enzymes (the peroxydase) is destroyed at the mentioned temperature. Mr. Barthel had now found that an examination of one of the other normal enzymes might be used for detecting an undue amount of bacteria in the milk. To use the usual bacteriological examination methods was not feasible when a large number of samples had to be examined. But his method was very simple and took, at the utmost, three hours to carry out. If methylene blue (free from formalin) is added to a sample of milk, a decoloration sets in as a result of the action of a reducing enzyme found in the micro-organisms of the milk. This decoloration is the more rapid the larger the number of microbes is. Mr. Barthel's method is as follows: To 10 c.c. milk 0·5 c.c. methylene blue solution is added. On the top of the mixture 2 c.c. paraffinum liquidum are poured in order to exclude the air. Place the test tube in a water-bath of 40–45° C. and note the time necessary for decoloration. If decoloration occurs within a few minutes, the milk contains certainly about 100,000,000 of microbes per c.c. and ought not to be offered for sale. Even in those cases where decoloration only sets in after an hour or so the milk is not fit for food, at least not for infants. Milk in which decoloration occurs in from one to three hours is of second quality. If longer time is required for decoloration, the milk is of good commercial quality. Milk fresh from the cow requires ten to twelve hours for decoloration. In the following discussion Professor Medin said that he thought that the destruction of the milk enzymes had something to do with the causation of Barlow's disease, which occurred only in artificially-fed children. He wished to warn against the buddeization of milk, a method of sterilisation originated by the Danish engineer Budde in 1900. Budde adds hydrogen peroxide to the milk, thus undoubtedly securing a bactericidal effect. But the enzymes did not seem to tolerate this method of sterilisation better than boiling. The only case of Barlow's disease which Professor Medin had observed in his own practice was in a child fed exclusively on "buddeised" milk. Furthermore, of a series of babies fed on "buddeised" milk in the Infants' Hospital of Copenhagen, not fewer than six of eight had Barlow's disease, but recovered in a fortnight when they were given raw milk. As to the real cause of Barlow's disease, it could not be the destruction of the enzymes alone, as during the years 1886–96 not a single case of the disease was observed in the Children's Hospital of Stockholm, notwithstanding the fact that at that time nearly all the babies were fed exclusively on boiled milk. Dr. E. Landergren drew attention to the fact that Barlow's disease had only been observed during the last ten years or so. In this period nearly all the milk on the market had been centrifuged and thus separated from the impurities and the leucocytes contained. In this way the milk was deprived of not only the greater part of its enzymes but also of a very important substance, viz. the lecithin, so that the boiled milk now contained far less lecithin than in former days. Perhaps this might contribute to the explanation of those disturbances in the nutrition which were nowadays observed in infants exclusively fed on boiled milk.—*Med. Record*, vol. lxxiv, No. 7.

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## A CLINICAL LECTURE

ON

# GASTRIC ULCER.

Delivered at St. Bartholomew's Hospital.


By W. P. HERRINGHAM, M.D., F.R.C.P.
Physician to the Hospital.


GENTLEMEN,—In the spring of this year there was admitted into this hospital a very pale ill-nourished girl, called Eliza R—, æt. 16 years, and she occupied No. 19 in Mary. The story she gave us was that she had never been really very strong, that she had had her catamenia every fortnight, but had had no serious illness since scarlet fever when ten years, and measles when two years of age. For three months she had had amenorrhœa, as we found on inquiry, but there was no sign of pregnancy. It was pure amenorrhœa from ill-health. She said that for the last three months she had had first of all pain, which began by being general, as she described it, all over her limbs and trunk. But after that it was principally upon her left side, the left hypochondrium and left loin. Further, we found that it came on very soon—she said immediately—after her food, that it lasted for several hours or until she was sick, that it was relieved by being sick. And another thing about it was that it was just the same whatever kind of food she took; even the lightest possible food produced pain quite as much as meat did. The next point about her symptoms was that she began to vomit soon after the pain began, and had continued to vomit ever since. The vomiting was produced by pain, it happened every day after nearly all her meals, and the contents of the vomit were simply what she had taken to eat, up till two days before admission, which was on May 13th. That night she twice brought up large quantities of blood, making a total quantity brought up of at least a quart. That was the history we

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had of her illness. When admitted she was seen to be a very ill-nourished girl, as I have said; she looked very pale, her heart and lungs were quite natural, and the blood showed the ordinary changes which you find after hæmorrhage, that is to say, diminution in the number of red cells and diminution in the hæmoglobin; but there was no increase of the white cells, and no sign of her having any blood disease except such as is secondary to hæmorrhage. On examining her we found that her heart and lungs were perfectly natural, that her abdomen was natural, except that she had a tender spot on the left side, just outside her rectus muscle in the left hypochondrium. The bowels were not opened until we gave her an enema, and we found that the fæces were rather abnormal in colour. They were dark, and on examining them with guaiacum and ozonic ether they gave the blue colour characteristic of hæmoglobin. So she was passing blood by the stools as well. We never saw any vomiting at all. So we could not prove she had had hæmatemesis apart from the history, but this fact of having melæna was sufficient proof of her story being correct. I think any of you would make at once, without difficulty, the same diagnosis which was made at the time, that of gastric ulcer. And in a case like that you may say, I suppose, that gastric ulcer is an easy diagnosis. But on the whole I should say the diagnosis, to be certain a patient has gastric ulcer or has not, is almost the most difficult thing in the whole of medicine. And it was very well said the other day at a meeting that the increase of our knowledge of late years, so far from having rendered the diagnosis of gastric ulcer more easy, has rendered it more difficult than it was before. To show you that, just take the symptoms one by one. In the first place, the pain. Pain occurs, you may say, in all diseases of the stomach, whether there is a breach of surface or not. And I should say that probably those diseases of the stomach which we call neuroses because they do not show any breach or alteration in the naked-eye appearance of the organ, cause quite as much pain as does gastric ulcer. Here is a case which happened many years ago now, and I have notes of it. It was in 1897. The patient was a girl named Maggie D—, æt. 27 years, a servant. She was admitted on May 25th. Nine years ago she had been in Charing Cross Hospital with hæmatemesis and the diagnosis was gastric ulcer. Five years ago she had been in Middlesex Hospital with hæmatemesis with the diagnosis of gastric ulcer. Eighteen months ago she had been in Hampstead Infirmary with hæmatemesis, and this time with melæna also, and again the diagnosis was gastric ulcer. Since January, 1897, she had been getting ill again, had had pain after her food, and five weeks before admission she had had a sudden intense pain, so intense that she fainted. When she recovered consciousness she found herself in an infirmary, she said. The pain was repeated. Ever since that attack of pain, that is to say five weeks before admission, she had been vomiting all the time, and had had another attack of intense pain, which led to her admission to the hospital. There was a consultation held about her, and it was decided that if she had not perforated gastric ulcer, at any rate she was in such a dangerous condition that the safest thing was to make an exploratory incision. Accordingly laparotomy was done, but there was nothing found when the abdomen was opened; the stomach was carefully examined back and front, handled carefully, and as much of it extracted from the external wound as possible. But there was no sign of disease, either inside or outside—no adhesions, no thickening. So the stomach was simply put back into the abdomen and the wound sewn up again. That patient did not have another symptom afterwards; all the pain went. There was no vomiting and nothing happened. Apparently she was cured by the stomach being taken out and handled. You cannot suppose that a case like that had a gastric ulcer; it would have been made worse by such treatment. Apparently there was no organic disease in the stomach, and yet her pain was sufficient to cause her on two occasions to faint. And then it is not only diseases of the stomach itself, whether organic or neurotic, which cause pain there; I suppose there is no pain much more intense than a certain form of gastralgia which is not due to disease of the stomach at all, but to a central disease, namely locomotor ataxy, in its gastric crises. They are sufficiently painful for people to make mistakes about them, and to cause the practitioner to believe that the patient is suffering from biliary colic. I have twice seen patients admitted into this hospital who bore scars of operations which had been per-

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formed for supposed biliary colic, the truth being that they had nothing whatever but the gastric crises of tabes. So there is a disease which is just as painful as ulcerous disease of the stomach which is not due to disease of that organ at all, but due to a central affection of the spinal cord. On the other hand, a great many cases of gastric ulcer cause no pain at all; many of the cases which come in here perforated have no history of pain until the perforation takes place. Sometimes the same is true of cases of violent hæmatemesis, but more rarely. It is the cases which perforate that are latent. I think that is a broad general rule. And so, as far as pain goes, not only can you be deceived by the pain occurring in other conditions which are not gastric ulcer, but also by the fact that the pain is not a necessary symptom of gastric ulcer itself. Still, when you find repeated pain occurring after food and continued for a considerable time, and to a severe degree, it is some evidence when you take it with other things, that the patient is suffering from gastric ulcer. And then take the second point, the symptom which goes with the pain, namely tenderness. I think that is very important. It is not the diffuse tenderness which you may find in any case of inflammation of the stomach. A person who upsets himself by eating indigestible food or taking anything unwholesome will get a general tenderness over the stomach very likely. But the tenderness which is characteristic of gastric ulcer is a tenderness localised to one spot in the stomach. And the best way to find it is to palpate with one finger, just as you do for the tender spot of appendicitis, pressing with the point of the finger over the epigastrium and hypochondrium, especially on the left side, and you will often find a definite area not bigger than your finger-tip perhaps, in which the patient complains of decided tenderness, whereas the neighbouring parts do not show any tenderness at all. And that is not only from the abdomen, but a similar spot you will often find behind and alongside the spinal column, usually half an inch or an inch to the left. That tender spot, I think, is a very important sign in the diagnosis of gastric ulcer. And there is another thing about the tender spot. You may find a tender spot in some cases of pain in the stomach, but it will shift about from day to day. We had such a case in the ward just a week or two ago, and most of those cases in which the tender spot shifts about you may safely say are not definite gastric ulcer, but something else, which is usually called neurosis of the stomach. Then comes the symptom of vomiting, and that, I suppose, is the commonest symptom in the whole of medicine. There is no disease which may not present it. All the infectious fevers may begin with vomiting, for instance, and I suppose it means that the nerves which pass to the stomach—that is, the vagus and the splanchnics, are more easily affected than those of other organs, and are more commonly the subject of reflex action. The characteristic of vomiting which is due to gastric disease is, of course, that it is repeated continually and occurs after food, that it is definitely due to what is taken into the stomach. But that is not proof of organic disease of the stomach. There is no vomiting which is so obstinate and so continually repeated after food as in those cases which are generally called hysterical or nervous. Nervous vomiting is a rare disease, but I have had one case under me in the last two or three years, and I have seen two or three others here in this hospital. I mean by it a condition in which you can exclude organic disease, not only of the stomach, but of any other part, partly from the absence of certain symptoms and partly from the presence of others. Such vomiting is not accompanied by blood, and hardly by any pain; there is no tenderness, or if there is it is a shifting tenderness and the patient is not severely ill. And then these nervous cases of vomiting are cases in which the vomiting is apparently remarkably easy—there is no distress caused by the effort to vomit. Indeed, they are cases in which the patient seems to like to vomit after her food, and the size of the meal or the nature of the food appears to make no difference whatever to the vomiting. The case I had was that of a girl, æt. about 19 or 20 years, who was a poor man's daughter. She had been in the habit of vomiting for the last three or four years repeatedly. She had been spoilt at her own home, had been kept in bed, and generally treated as an interesting person. I felt confident, after watching her, that she wished to vomit and would not try to stop it. Her stomach had got into the habit of it, and we could not break her of it, and she went out as bad as she came in, in spite of every change in diet that we could think of. The only thing which stopped the habit for a time was some painful

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impression which we produced upon her which we made to follow as a consequence of her vomiting. I think it was some saline solution injected subcutaneously whenever she vomited. And that for a few days made a decided impression and stopped the vomiting But it soon started again and we were powerless to stop it. She was as well in her ordinary health as you or I. That is a case in which vomiting is not proof of organic disease of the stomach at all. There are two points about vomiting which are, to a certain extent, in favour of gastric ulcer as the cause of the vomiting. One is the presence of free hydrochloric acid, even perhaps an excess of hydrochloric acid in the vomited matter or in the contents of the stomach withdrawn experimentally. And secondly, the presence of blood in the vomit. As regards the presence of the acid in the vomited matter or in the contents of the stomach, it is generally known that cases of gastric ulcer often do show a considerable amount of free hydrochloric acid. In this particular case this girl vomited not at all after she came in, and seeing she had had severe hæmatemesis I did not like to put in a gastric tube and withdraw the stomach contents—I did not feel justified in doing so. So we remained without any evidence of acidity in her stomach contents until about a month after she came in. On June 16th we thought we might safely extract the contents of the stomach with a test meal, and this we did. That showed the entire absence of any free acid at all. That is at first sight rather startling, for a reason which I will tell you in a minute. It rather upsets your diagnosis and leaves you puzzled as to whether it is due to ulcer or is another form of hæmatemesis, of which I shall speak. But when you think of it it is quite easily explained because it is well known that any wasting or exhausting disease, or almost any condition of debility, is sufficient to stop the secretion of hydrochloric acid in the gastric juice, so that a person only requires to be a little out of health for the secretion of hydrochloric acid to be stopped. This girl, at the time I tested her, had been suffering from the hæmorrhage of gastric ulcer, her hæmoglobin was still very deficient, and she had been kept in bed on a light diet for a whole month, so she was in a weakly condition, and I think that was enough to account for the fact that we got no hydrochloric acid n the stomach contents. The second point concerns the occurrence of blood in the vomit, and there we come upon another very difficult question. It seems to be allowed by almost everybody that hæmatemesis is not always a sign of breach of surface of the stomach. It has been described even in hysterical vomiting. And by that I mean blood in large quantity. Charcot describes hæmatemesis in large quantities as a result of hysterical vomiting; but this I am inclined to doubt. But we know there must be very many cases occurring in young women at the typical age for an ulcer of the stomach, which, judging by the discrepancy between post-mortem statistics and the statistics during life, do not depend upon gastric ulcer. One can make some estimate of the number of cases which are seen with scars or evidence of gastric ulcer amongst women, and we find that they bear a certain proportion to the cases occurring amongst men. But that proportion is not anything like the proportion which cases of gastric hæmorrhage amongst young women bear to similar cases among men. There must be something else, some other condition; some people have said it is due to erosions of the stomach. Others have talked about bleeding points in the organ, and some others have contented themselves with saying it must be like cases of epistaxis occurring in childhood. When you see a boy with his nose bleeding you do not think he has an erosion of the mucous membrane of his nose; he apparently bleeds from mere congestion, and I believe it is so in many cases of hæmorrhage from the stomach in women. Can you draw any line of distinction between those cases of hæmorrhage which occur in ulcer and those in which you think the hæmorrhage is due to something else? It has been suggested that the absence of acidity will be against, and the presence of acidity will be in favour of, there being ulceration. But such a case as this one would leave you in doubt. Here you have a case in which the contents of the stomach are not acid but alkaline, and if you relied upon that test without considering the reasons for it, and without considering the circumstances in which it might vary, it might land you in a difficulty in diagnosis. When there is hæmorrhage from the stomach in quantity anything over a pint, I think you are generally safe in thinking that it is due to ulceration of the stomach if there is also some other symptom pointing to it, such as pain. But the mere fact

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of the hæmatemesis itself, however severe, is not sufficient ground. There have been three cases in the last twenty years in this hospital in which people have died of hæmatemesis, and yet nothing whatever has been found wrong in the stomach. One occurred here in 1888 in Dr. Andrew's ward. A young man called S—, æt. 26 years, died of hæmatemesis, sudden and profuse bleeding, and nothing whatever in his stomach or intestines was found to account for it. I saw, in Dr. Gee's ward, a girl named Rose D—, æt. 17 years, who was chlorotic and who died with profuse hæmatemesis. Post mortem, blood was found in the stomach, and the intestines were full of blood. There was no sign of where the blood came from and there was no form of abnormal anæmia, such as pernicious or splenic, which sometimes cause profuse hæmorrhage. And only the other day a case was under Dr. Ormerod with profuse hæmorrhage from the stomach. He died, and nothing whatever could be found to account for it. So I ask you to remember that sometimes profuse hæmatemesis occurs without any visible breach of surface whatever. But as a general rule if there is severe hæmatemesis and other symptoms as well, you can fairly well distinguish those cases as instances of gastic ulcer from other cases of slighter disease of the stomach. The cases of slighter disease of the stomach often cause plenty of pain and vomiting but the hæmatemesis is slight. Therefore it comes to this, you see that there is nothing pathognomonic about the symptoms of gastric ulcer, there is no one symptom which you can put your finger on and say if the patient has that symptom she has gastric ulcer. You have in each case to make out a combination of symptoms, and in each case you have to exercise your judgment as to the general aspect of the patient and the general run of symptoms, and make your diagnosis from the probability in the individual case. And, as I told you at the beginning, there is not a condition in medicine in which the diagnosis is so uncertain. I will mention another point in regard to which this case was interesting. Whatever your diagnosis of a case is, if you are in any doubt about it being gastric ulcer, the right thing to do is to treat it for the severest disease which it possibly may be, namely, ulcer. And the treatment of those cases of ulcer is now generally starvation for a short period. When I was your age and was a student, that was never dreamed of. These people used to have milk, or milk and whey, or milk and soda-water, or milk and lime-water, from the first, and I am not at all sure that they did very much worse for it. I think we have taken it rather for granted that the stomach will not tolerate any food at all. However, at the present day I should say that very few people feed gastric ulcer patients by the mouth. Most people starve them for a few days, and give them what nourishment they think is necessary, in the form of nutrient enemata. These vary in composition, either eggs, or milk, or meat extract, or peptone or something of that sort, very often pancreatised, and generally with a little opium added to prevent irritation of the bowel. These are given every three or four hours, and every day the intestines are washed out, so that any accumulation of undigested or absorbed material should be removed. I have always doubted whether that was of any real benefit to the patient, and especially since I saw a case in my own family, that of a doctor, who was treated in that way for about ten days. He told me that the washings out, although carefully done—and he was just as careful in seeing that they were properly done as I was—were never thoroughly satisfactory. He did not believe that they removed all the material that he had not absorbed. About ten days after being first taken ill he passed an enormous evacuation of the most disgustingly putrid material you can imagine. It nearly made him sick, and it quite made the nurse sick. And he said: "If I was retaining all that in my intestine for that length of time, it is impossible that the treatment can have done me much good." And I agreed with him. In the second place, it is a very unpleasant method of treatment, and it is very irritating to the bowel in some patients. And in the third place, I have always doubted whether the absorption of nutriment really took place to any appreciable extent. In the last year or two a good many experiments have been made to test that. The treatment was originally adopted in consequence of experiments made upon animals, dogs chiefly, by injecting various forms of nutrient enemata and testing the excretions afterwards. It has lately been tested in man much more carefully than it was before, and with the result that those people who have made the experiments, such as Dr. Boyd and Dr. Robertson in Edinburgh, and Dr. Edsall and Dr. Miller in

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the United States, and one or two German observers, have been extremely doubtful whether any more than the minutest amount of nitrogenous material or of fatty material, or even of sugar, the easiest absorbed of all, was taken up by the intestinal mucous membrane. And if it is not, if there is very little absorbed, it surely is not worth while to put patients and nurses to the trouble and inconvenience of such a treatment as that. And in consequence for several years I have occasionally used simple saline enemata, as I used in this case. We gave this woman five pints of simple saline injection every day for five days, and her weight during those five days did not alter in the least; for on May 15th it was 6 st. 1½ lb., and on May 20th it was 6 st. 1½ lb. On the 21st and 22nd nutrient enemata were given, because Dr. Langdon Brown was making experiments on this point then, and on the 23rd she began to have a little by the mouth, but still with three pints of saline solution by the rectum. From that day we gradually increased the quantity of food given by the mouth, and on June 12th she went on to ordinary diet, with chicken. On June 19th her weight was 5 st. 10 lb. So she has almost regained her natural weight. Her blood condition improved from the beginning, and she recovered without the slightest drawback. That is about the third or fourth case in which I have used these saline injections without any nutrient enemata at all. The patient immediately before this actually gained weight under the saline enemata. And the patient before that again lost, I think, one pound in six days. So that practically under saline injections, five pints daily, the patient does not lose weight. Of course they excrete a certain amount of urea, which is at the expense of their tissues, but as far as I can tell at present it does not seem to be much more at the expense of their tissues than it is when you give them nutrient enemata. So I do not think there is any great advantage to be obtained, and I know there is great inconvenience in the ordinary treatment by nutrient enemata which is commonly practised.

*September 14th*, 1908.

# A RESUMÉ OF RECENT CLINICAL STUDIES OF ATHEROMA AND ARTERIO-SCLEROSIS.*

By GEORGE OLIVER, M.D., F.R.C.P.Lond.

(*Concluded from p.* 342.)

(2) THE RELATION OF ATHEROMA AND ARTERIO-SCLEROSIS TO ARTERIAL BLOOD-PRESSURE.

Thickening of the arterial wall introduces an abnormal condition into the determination of the arterial pressure in man; for some of our modern methods—and notably that of the armlet—are founded on experimental work mainly on normal subjects in whom the arterial wall is soft and non-resistant.

*The Armlet trustworthy as a Physiological Method.*

The results thus obtained have been perfectly consistent; and in animals the extra-vascular application of the air-bag has even been shown to yield readings identical with those of the intra-vascular method of determining the arterial pressure. Therefore, as a physiological method the armlet is quite satisfactory.

*Is the Armlet equally trustworthy when the Arteries are Sclerosed?*

But in clinical work we frequently find that the arterial wall is thickened by atheroma and arterio-sclerosis; and we have now to consider what is the effect of that thickening on the data furnished by the armlet method? Experimental work on isolated sclerosed arteries having indicated that they are closed by a comparatively nominal increase of pressure (*e. g.* 5 mm. Hg.)† it was natural that observers should infer that thickening of the arterial wall should not materially vitiate the determinations of the armlet—that the readings furnished by it should be practically accurate measurements of the arterial blood-pressure. Moreover, this inference was strengthened by the not infrequent observation of low armlet readings

* A Lecture delivered at the Medical Graduates' College and Polyclinic, London, 1908.

† 'Brit. Med. Journ.,' 1905, p. 866.

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derived from arms in which the brachial artery was considerably thickened or even calcified.

*Very high Armlet Readings in Arterio-Sclerosis are in themselves Suspicions of Error.*

But the very high determinations furnished by the armlet in cases of diffuse arterio-sclerosis, whether associated with renal disease or not, such as the systolic readings of 250 to 300 mm. Hg. or even higher, naturally prompt one to inquire—do these figures represent the actual arterial pressure *plus* the extra resistance required to close the thickened artery? Dr. William Russell,* who criticises our acceptance of the armlet readings in arterio-sclerosis as blood-pressure determinations, regards the high readings obtained as inconsistent with our physiological knowledge; and my own observations elsewhere published† afford some support to his contention.

*Disparity between the Digital and the Armlet Estimation of the Blood-pressure.*

In practice you will not infrequently meet with cases of atheromatous thickening of the accessible arteries in which you will find a comparatively soft, or quite soft, radial pulse, though the brachial reading obtained by the armlet may be distinctly super-normal. In such cases in our early observations with the armlet we were inclined to suspect the accuracy of the finger rather than the armlet. But later experience has shown that this aspersion on the tactile method was ill founded. In many cases in which this disparity was apparent I have been able to prove to my own satisfaction by another method of measuring the arterial blood-pressure, and by the collateral clinical evidence, that the estimate of the pressure afforded by the finger was much nearer the truth than that furnished by the armlet. All such cases illustrate the conclusion that the armlet readings are apt to be raised, and may be very considerably raised, by the additional pressure required to *completely* close a thickened artery; and that the figures thus obtained are not always determinations of the actual blood-pressure. I mention this fact somewhat pointedly lest now and then you should be misled by sole reliance on the armlet reading; for you should collate it with all the available data in each case.

*In Atheroma and Arterio-Sclerosis the Armlet Readings may vary in different Areas.*

The position, that thickening of the arterior wall vitiates the armlet readings as determinations of the blood-pressure, is supported by a fact which is the outcome of some recent observations (elsewhere published*) I have made on cases of atheroma and arterio-sclerosis, namely, the fact that in such cases the armlet is apt to furnish different readings derived from the two arms or the two forearms, and more particularly from the arm and forearm of the same side. You will doubtless be aware that the armlet readings are uniform in these different areas when the arterial wall is normal. Therefore the fact of disparity in the pressure readings in the same subject can only be explained by the varying thickesses of the arterial wall. It cannot be due to actual differences in the arterial blood-pressure.

*The Clinical Value of the Armlet Method.*

You should not, however, conclude from the foregoing facts that the value of the armlet method is much impaired. When the arteries of the arm are normal it is certainly the best method we possess; and when in any case its readings are uniform in the arms and forearms we have as a rule presumptive evidence of the integrity of the vessel wall in the limbs, but only presumptive evidence—for this matter can only be demonstrated by other objective evidence. I have found it useful in the study of the early stages of arterio-sclerosis, when digital examination of the arteries gives us merely equivocal evidence of thickening, or does not afford very distinctive evidence of it; and the corroboration it yields in the more advanced stages to the results of the objective examination of the vessels, though not always necessary, is yet sometimes acceptable. Moreover, even in all cases of arterial thickening the figures which it yields, when the armlet is always applied to the same area, are valuable in indicating variations of pressure; and in most cases it seems to me that the lowest reading obtainable is not generally misleading for all practical purposes as a blood-pressure determination.

* 'Arterial Hypertonus, Sclerosis, and Blood-pressure,' by W. Russell, M.D., December, 1907.

† "Studies in Blood-pressure," January, 1908.

* 'Studies in Blood-pressure,' pp. 119, 120.

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*Can the Arterial Pressure be read with approximate accuracy in Atheroma and Arterio-Sclerosis?*

I am personally satisfied that no mode of determining the arterial blood-pressure which depends on *closure* of the artery is quite trustworthy when the arterial wall is thickened. When that is the case the only methods which have afforded me satisfactory results in accord with clinical experience are those which are founded on the principle of balancing the pulse-pressure wave, *e.g.* by the hæmodynamometer. I have elsewhere * described this method of measuring the arterial pressure, which gives readings identical with those of the armlet when the arteries are normal, but are apt to be lower than these when the arteries are sclerosed. The following is a brief outline of my results in atheroma and arterio-sclerosis obtained by the adoption of the two methods:

(1) In many cases the hæmodynamometer readings were generally lower than the armlet determinations.

(2) Some armlet readings distinctly super-normal (*e.g.* 160–220 mm. Hg.) were fairly normal according to the hæmodynamometer (125–140).

(3) Some systolic armlet pressures comparatively low (*e.g.* 105–130 or 140) were read still lower (105–115) by the hæmodynamometer.

(4) Very high armlet readings (250–300) or over have rarely exceeded 200 mm. when determined by the hæmodynamometer.

*The Arterial Pressure in Atheroma: Normal or Low Readings.*

There is quite a prevalent notion that the arterial blood-pressure must rise if the arterial walls are thickened. There is nothing more fallacious; and a little clinical observation will soon satisfy you that there is a large percentage of our cases of arterial thickening in which the pressure is not only not at all raised but may be somewhat subnormal. Sir Clifford Allbutt and other observers have also recorded this fact. You will find that the large majority of these cases belong to the group of simple atheroma, or what has been termed involutionary arterio-sclerosis,† affecting the medium-sized vessels and not complicated with hypertonus or with diffuse arterio-sclerosis. According to my observation an apparently very extensive or widespread atheroma is quite consistent with a low blood-pressure. As a matter of fact the majority of elderly people are the subjects of this quiescent form of arterial thickening, and after many years the end may come through pneumonia, or some other cause not in any way connected with the state of the arteries. I have shown elsewhere * that it is quite usual to find it in those who attain to an advanced age—*e.g.* from 80 to 100 years; and in some such cases I have observed that the armlet may yield fairly high and different readings in the two arms and forearms, whereas the hæmo-dynamometer will show a lower arterial pressure, *identical on both sides and in all the accessible arteries*, and, indeed, a pressure similar to that which is usually observed in earlier life.

May it not be that in atheroma the blood-pressure remains, as a rule, normal, or may become somewhat subnormal because all the other involutionary changes are equably diminished? When, for example, the senile obsolescence of the peripheral vessels proceeds *pari passu* with reduction of the blood volume and with the diminished activity of all the physiological processes, the arterial pressure will likewise lessen. In fact, in normal senescence the arterial pressure tends to become somewhat subnormal. I have often thought there is a slight recrudescence of pressure in the elderly at the grand climacteric in man (from 55 to 65 or 70 years), which eventually subsides during the period of normal involution.

But atheroma is not the only form of arterial thickening in which normal or comparatively low readings of the arterial pressure are frequently met with. The same fact is common in the forms of arterio-sclerosis produced by syphilis, typhoid and other infective fevers, diabetes and some auto-intoxications.

There is, therefore, conclusive evidence to show that the arterial pressure very frequently does remain unaffected by thickening of the arterial wall; and in approaching the study of blood-pressure measurement in connection with atheroma and arterio-sclerosis it is important that you should disabuse your mind of the notion of a necessary association between arterio-thickening and heightened arterial pressure.

* 'Studies in Blood-pressure,' 1908.

† See Allbutt, 'Brit. Med. Journ.,' 1906, vol. i, p. 6.

* 'Studies in Blood-pressure,' 2nd edition, 1908.

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*Supernormal Arterial Pressure in Atheroma and Arterio-sclerosis.*

But in many cases, not only of arterio-sclerosis but of atheroma, instrumental observation indicates that the arterial pressure is raised—and in some cases raised very considerably; and it is a matter of no small practical importance to prove as far as you can that the readings thus obtained are actually hypernormal readings of the blood-pressure, and not merely hypernormal readings of arterial resistance. I have shown you that these two facts are not always identical when the arteries are thickened in certain cases. It is therefore important to make your conclusion as solid as possible by calling in the aid of the collateral clinical facts in each case; such as the evidence of the well-trained finger in estimating the pulse-pressure and all the available data derived from a thorough examination of the heart and the aorta. A rise in the arterial pressure cannot be long continued without making an impress on the ventricle and on the aorta—either developing hypertrophy or dilatation of the ventricle or elongation of the aortic arch. Hence the practical value in routine work in recognising the outward swing of the apex and the accentuation of the second aortic sound—either by auscultating in front or behind on the line between the left scapular angle and the seventh dorsal spine (after Friedmann).

The blood-pressure is supernormal in a large proportion of the cases of arterial thickening. In practice we find a large group of cases of sclerosed arteries in which the pressure is raised long before we can detect thickening in the accessible arteries; but I am disposed to think that these cases do not constitute the whole field. We know that arteries may become markedly sclerosed without any increase in the arterial pressure; but in a certain number of these cases I am persuaded that the pressure may eventually become persistently hypernormal. If so, we have a group of cases in which the lesion precedes for some time the rise of the pressure; but you must remember that such cases form a comparatively minor group.

I shall conclude this section of my lecture by tabulating the following clinical groups of cases which my pressure observations in arterio-sclerosis and atheroma have led me to formulate.

| | |
|---|---|
| I. Normal or lowered arterial pressure mainly. | (1) Atheroma (normal involution).<br>(2) Arterio-sclerosis of toxic origin (following infective fevers, syphilis, etc.) |
| II. Hyper-normal arterial pressure mainly. | (1) Hyperpiesis of Allbutt or pre-sclerosis of Huchard terminating in arterio-sclerosis.<br>(2) Atheroma associated with or terminating in arterio-sclerosis.<br>(3) Arterio-sclerosis in which fibroid degeneration is prominent (arterio-fibrosis or arterio-capillary fibrosis of Gull and Sutton and the angio-sclerosis of Thoma). |

In the last of these five groups we may presume that the arterio-sclerosis is the outcome of a general fibroid degeneration, which may likewise invade the kidney and other organs and tissues; and when the kidneys are involved the pressure readings are as a rule higher than in the other forms of arterio-sclerosis. To this group belong those cases described by Thoma under the designation of *angio-sclerosis*, in which the whole vascular system (the capillaries and veins as well as arteries) is invaded by sclerosis. In this group I have observed a great diminution of the response to a dose of the nitrites; whereas in atheroma and in the ordinary run of cases of arterio-sclerosis that response is frequently fairly good, and in atheroma may be quite normal. The normal response consists of a dilatation of the arterial calibre (shown by the arteriometer) and a fall (*e.g.* 20 to 30 mm. Hg.) in the arterial blood-pressure. In the fibrotic form of arterio-sclerosis I have also found that the radial calibre afforded by the arteriometer in the sitting position varies much less when the patient assumes the recumbent posture than it does in other forms of arterial thickening or in the normal condition of the arteries; in fact we may fail to find any evidence of postural variation in the radial calibre in arterio-fibrosis.

(3) Pathogenic Theories—Some Ætiological Factors.

Observers have been much exercised as to how arterial thickening is produced. I shall now briefly review some of the explanatory theories which have been suggested. Will the mere rise of the arterial pressure produce arterio-sclerosis? This *mechanical theory* assumes that the arterial

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pressure is raised by virtue of some alteration in the working of the circulatory mechanism, or in the blood, or in both; and the strain on the arteries thus induced suffices to cause the lesion, this being secondary to the rise of pressure. This view has been advocated more particularly by Traube, Huchard, Allbutt, and W. Russell, and is now held by the majority of clinicians and by many pathologists. Experimentation on animals has demonstrated that the arterial pressure when mechanically raised by compression of the abdominal aorta will cause the arterial lesion (Moritz, Harvey). Harvey, in the Cambridge laboratory, has "shown that merely compressing the aorta of rabbits with the fingers for two or three minutes daily, thereby raising the blood-pressure 30 or 40 mm. Hg., produces degeneration of the aorta above the point of compression without causing any change in the vessel below. No other effect was observed on any tissue, and the rabbits appeared normal in every other way. The degenerative changes so induced were exactly comparable with those induced by the injection of drugs."*

Dixon also observes: "Within the last two or three years it has been shown by different workers in this laboratory, that any drug which has the power of considerably raising blood-pressure will, when injected into the circulation of healthy animals, bring about degeneration of the middle coat of the arteries. . . It has been shown that digitalis, squill, apocynum, barium, lead, adrenalin, nicotine, and even inhalation of tobacco smoke will bring about these changes."† These observations suggest, according to Dixon, the *modus operandi* of violent exercise in producing arterio-sclerosis, especially in the elderly, in whom the blood-pressure rises more from exercise than in the young. Moreover, observation shows that work, and especially overwork, thickens the arteries; we frequently see, for example, that in a part most exercised, such as the arms and forearms, the arteries become more sclerosed than elsewhere, or those arteries are alone thickened; and in such cases, as a rule, the right radial is more sclerosed than the left (unless the subject is left-handed). In the same way we often observe that the temporals are frequently thickened and curling in those engaged much in intellectual work, or in those who are much worried and anxious. If you examine the arteries of a number of young working men you will be surprised at the high percentage of cases of palpably thickened arteries. In such cases, however, the thickening is generally perfectly smooth and uniform, and is quite unlike that of atheroma and arterio-sclerosis. I am persuaded that it is due to hypertrophy of the media, and is the normal effect of continuous muscular work, and you should be on your guard not to accept it as evidence of arterio-sclerosis. But I am persuaded that persistent hard physical labour does favour the earlier appearance (by perhaps from ten to twenty years) of atheroma and arterio-sclerosis in those following laborious employments, such as navvies, labourers, porters, etc., than in those following less physically arduous occupations. Of course in such cases there are generally other factors at work, such as underfeeding, the influence of exposure, alcoholic excess, and other adverse conditions of a hard and strenuous life.

Huchard has for at least twenty-five years been the persistent advocate of the hypertonic theory, which implies generally a rise in the arterial pressure in the aorta and large arteries. He holds that toxic substances derived from food in excess or in noxious quality are absorbed into the blood and excite spasm of the arterial system; and he believes that this condition of angio-spasm, denominated by him the "pre-sclerotic period," may exist for some time without developing arterio-sclerosis when the hypertension is amenable to control or cure by hypotensive treatment. Ultimately the vessel spasm produces the lesion, when, however, treatment is less effective or is of no avail.*

Dr. W. Russell is a prominent advocate of this view. He says: "My contention is that certain substances present in the blood, even in small quantity, cause arterial and capillary contraction. This is the fundamental fact, and will be found to be the first step in all raising of blood-pressure, which goes on to arterio-sclerosis."†

Without subscribing to Huchard's views, Sir Clifford Allbutt has graphically described under the term "hyperpiesis" a condition of persistent and

* Prof. Dixon on "Arterio-sclerosis," 'Proc. Roy. Soc. Med.,' vol. i, No. 4.

† Dixon, *op. cit.*

* "Traitement de la Présclérose," 'Bull. de l'Académie de Médicine,' January, 1907.

† *Op. cit.*, p. 37.

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excessive arterial pressure caused by increased peripheral resistance and producing secondary arterio-sclerosis which first begins in the aorta.* The best examples of this group of cases are met with among the over-fed, or in those suffering from mal-alimentation as well as super-alimentation, and in the gouty. I see a fair number of these cases and I have frequently confirmed Sir Clifford's favourable opinion as to the effect of treatment when applied early or before the arteries become sclerosed.

The pressure theory of causation seems to fail us, however, in those cases of arterial thickening in which we have no evidence of hypertension or of supernormal aortic pressure—such as many cases of involutionary arterio-sclerosis and of the infective or toxic forms of the disease generated under a hypotonic condition of the circulation. To account for the lesion in such cases we must invoke some other explanation than the mechanical one.

The *toxic theory* has many facts (experimental and clinical) to support it. It is believed—indeed, it is an accepted fact—that many toxins may affect the arterial wall independently of their power to raise the arterial pressure. For example, it has been shown that the exogenetic infections of acute rheumatism, scarlatina, variola, diphtheria, typhoid fever, measles, influenza, tuberculosis, malaria, and syphilis, will produce arterio-sclerosis. In such cases endarteritis in the first instance may be set up, which extends to the media. Then, again, experiment has shown that bacterial agents, such as *Bacterium coli* and staphylococci will produce the lesion.

Just now perhaps the most interesting aspect of auto-intoxication is the suggested production of arterial disease from putrefaction in the large intestine, which is regarded by Metchnikoff as the cause of senescence and of atheroma. This question, it should be noted, is not one of mere academic interest, but is of some practical importance if experience should prove that Metchnikoff has furnished us with a remedy, namely, the colonisation of a pure culture of the *Bacillus lacticus* (the Bulgarian variety) in the bowel.

This investigator asserts that the bacillus taken by the mouth passes through the stomach unchanged and develops the lactic acid fermentation in the bowels, where it supplants the putrescent bacilli. In this way he believes that intestinal auto-intoxication is more effectually rectified than by the administration of antiseptics and purgatives. According to him this colonisation of the master bacillus is effected, either by administering a lacto-bacilline tablet after each meal containing sugary matter, or by giving skimmed milk curdled by the lacto-bacilline powder.* It may be interesting in this connection to refer to a sentence in Harvey's report of the post-mortem examination of Thomas Parr, to the effect that Parr's ordinary diet consisted of sub-rancid cheese, milk in every form, coarse hard bread, and small drink, generally sour whey. "On this sorry fare, but living in his home free from care, did this poor man attain to such length of days." But on his removal by the Earl of Arundel to London shortly before his death his diet was enrichened; "whence it came that, by day or night, observing no rules or regular times for eating, he was ready to discuss any kind of eatable that was at hand," and Harvey thinks that this departure from his long accustomed simple fare with its sour whey in the country precipitated the end.

When the arterial lesion is produced by toxic substances—such as lead, barium, ergot, tobacco, and adrenalin—which raise the blood-pressure, it is difficult to say whether it arises from the increased pressure or from the action of the toxin on the arterial wall, or from both these causes.

The study of the *supra-renal origin* of arterio-sclerosis has been assiduously prosecuted during the past few years, with results which point to the important *rôle* it may play in the pathogenesis of the disease. A large number of experimentalists in France, Germany, Italy and America, following Josué in France, have demonstrated that lesions indistinguishable from atheroma are produced in animals by the intra-venous injection of adrenalin, the lesions involving the aorta and occasionally other large arteries (*e. g.* carotids and iliacs) as well as visceral arteries (renal, mesenteric and pulmonary); and several pathologists have observed considerable hypertrophy of the supra-renal capsules and of the accessory supra-renal glands in a large proportion of autopsies in the atheromatous and the aged, and the frequency of supra-renal

* 'Brit. Med. Journ.,' 1907, vol. i, p. 5, and several articles in the journals of previous years.

* 'These preparations are sold by La Societé "Le Ferment," 77, rue Denfert-Rochereau, Paris.

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adenomata in interstitial nephritis. But to discuss the evidence in favour of this interesting aspect of the pathogeny of arterio-sclerosis and the various objections which have been raised to its adoption would require more time than can be afforded in this lecture.

But apart from the question of the causal relation between the over-secretion of adrenalin and arterial thickening, may not the inadequate production of the hypotensive secretion of the testis, ovary, and thyroid play a similar *rôle*? Hence it may be that the retention of sexual life and virility may delay or prevent the development of atheroma.

Though experimentalists have likewise demonstrated that *nicotine* produces in animals the same arterial lesion as adrenalin, I cannot say that my clinical observations have as yet afforded me any cogent evidence to show that tobacco smoking is an important ætiological factor in atheroma or arterio-sclerosis. As Professor Dixon points out, it may be that those who smoke habitually become immunised to nicotine by the production of a ferment which is generated in the liver, so that the occasional cigar may prove to be more injurious than the regular smoke which maintains immunisation, and perhaps smoking may be more pernicious when the habit is contracted late in life than when it has been indulged in from an earlier age. So far as my own observations go I cannot say that it has struck me that arterio-sclerosis is more developed in habitual smokers than in non-smokers; and in some great smokers whom I know I have failed to discover any evidence of the disease.

Alcohol is generally regarded as an important cause of arterio-sclerosis; and no doubt it is so in those who drink habitually and freely of spirits or malt liquors, and it is a fact, as pointed out by Dickinson, that those engaged in the liquor trade are, on the whole, more liable to arterial disease than others. But my clinical observation does not allow me to attach much weight to the causal influence of the moderate use of alcoholic beverages. There is no doubt, however, that the subjects of arterio-sclerosis are better in every way without alcohol in any form, or tobacco.

But there is a more potent cause of arterio-sclerosis than tobacco and alcohol, which may, moreover, find insidious entry into the system, and that is *lead*. Recent French writers seem to me to be more keenly alive to this evil than we are. Putnam has discovered lead in the urine in 17 per cent. of healthy subjects; and according to Gautier each Frenchman absorbs on an average a demi-milligramme of lead per day. Roget and Gouget believe that a good number of the cases of arterio-sclerosis in which the cause escapes us are due to insensible but continued saturnine intoxication.* The practitioner should keep a sharp look-out for this hidden enemy, especially when the water supply is soft and derived from peaty soil; and he will now and then discover the trail of the poison in the arteries and the blood, for the lowered hæmoglobin content is a valuable tell-tale of plumbism as well as the sclerosed arterial wall.

*Metabolic theory.*—But apart from an increment of the arterial pressure or the presence of toxic agents in the blood, some writers believe that arterio-sclerosis may result from trophic or metabolic causes. And in support of this view we have the fact that it is commonly met with in those who are predisposed to trophic troubles in those tissues which are either non-vascular or are sparsely supplied with blood-vessels, such as the cartilages, nails, cornea, lens. Moreover, as pointing in the same direction we have the observations of Trunecek, showing that in arterio-sclerosis the salts of the serum and of the tissues are deficient, and that the subcutaneous injection of them ameliorates or removes symptoms associated with the sclerosis. Furthermore, Levy, of Paris, and some other observers have found that the oral administration of these salts—which have been given in tablet form—is equally beneficial.

### *Some other Ætiological Data.*

I shall now adduce a few brief notes on the influence of age, sex, temperature and alimentation on the production of arterial thickening. Senescence disposes to the loss of elasticity and to the hardening of the tissues of the body; and according to Boy-Teisseur it produces in the arteries proliferation of the connective tissue in the three tunics. Though atheroma is common in the old we should remember, as I have pointed out, that it is not a necessary result of senescence. It is perhaps doubtful if we can strictly regard age *per se* as an important factor in the production of arterio-sclerosis; for what we see developed in the course of time is probably only but the result of the operation of other causes, time being merely the revealer. The age incidence of arterial thickening and degeneration varies considerably, not only in individuals but in families, some degenerating quite early, others quite late, and all illustrating the oft-quoted aphorism, "a man is as old as his arteries." Arthritic subjects (gouty or rheumatic) are as a rule disposed to early arterial degeneration. Arterial thickening is more frequently met with in men than in women before

* Roget et Gouget, "Nouveau Traite de Médicine et de Thérapeutique de Brouardel," Gilbert et Thoinot, fasc. xxiv.

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the menopause; repeated pregnancies and the menopause are, however, predisposing causes, and an arduous life induces arterio-sclerosis in women just as it does in men.

The influence of temperature is an ætiological factor; for example, prolonged exposure to a cold moist atmosphere may be an important cause (among others) of the progressive obliterative arterio-sclerosis seen among Russian Jews, and of the comparatively early appearance of arterial thickening in those much exposed to varying temperatures, such as agricultural labourers.

The food factor in favouring or retarding the development of arterio-sclerosis has not been elucidated by experimental work. There is, however, a general belief that an excess of animal food may predispose to arterio-sclerosis, whereas the adoption of a vegetable diet may have a favourable effect in preventing it. But this conclusion does not seem to be quite supported by the following facts: that arterio-sclerosis is rarely seen in animals except in very old horses and oxen, and occasionally in rabbits, and that calcification is fairly common in herbivorous animals, whereas in the dog arterio-sclerosis is quite rare, Thoma having encountered it only once in a dog, and that dog was seventeen years old.* Therefore, from the mere food standpoint it would seem that the flesh-feeding animal—the dog—is less disposed to arterio-sclerosis than herbivorous animals. We sometimes meet with cases of arterial thickening associated with sub-alimentation and low metabolism, in which I think we are justified in prescribing meat somewhat liberally, providing the kidneys are sound and the arterial pressure is not raised. In by far the majority of cases of arterio-sclerosis, however, experience is in favour of a diet chiefly vegetarian or lacto-vegetarian, in a word, a diet containing a small proportion of meat proteid with an ample supply of green and other vegetables and vegetable-derived products with fruits.

*Resumé of Causation.*

On briefly reviewing the ætiological factors of arterio-sclerosis we may conclude:

(1) That it is misleading to attempt to refer it to any one cause in all cases.

(2) That the causes are as a rule multiple, though a certain cause, or more than one cause, may be predominant in individual cases.

(3) That the leading causes are: (*a*) persistent supernormal arterial pressure, however induced; and (*b*) toxins of various kinds, either generated within the body (endogenetic) or introduced from without (exogenetic).

(4) That in addition to these determining causes there are certain predisposing factors, such as heredity and trophic proclivities.

*September 14th*, 1908.

* Gouget, *op. cit.*, p. 21.

## WITH MR. R. H. JOCELYN SWAN IN THE OUT-PATIENT DEPARTMENT OF THE CANCER HOSPITAL.

DURING the course of an afternoon's out-patient clinique the following cases were examined and commented upon by Mr. Swan:

CASE 1. *Carcinoma of the breast.*—Woman, æt. 38 years, came to the hospital complaining of a lump in her right breast which she had noticed for about a month. At first she disregarded it as it caused no pain, but during the last few days she had experienced slight shooting pains in the breast. On examination the breast was well developed and in the upper and inner quadrant a small, rounded, firm, ill-defined tumour could be felt. It was freely movable on the deeper tissues, but not in the breast tissue; the nipple was not retracted, but on making slight pressure in the space between the tumour and the nipple the latter became slightly drawn over towards the mass. There was no sense of fluctuation in the tumour and no glands could be felt enlarged in the axilla or clavicular spaces. No other tumour could be felt in the same or in the other breast.

Mr. Swan pointed out that practically the only conditions giving rise to these symptoms would be a tense cyst or an early carcinoma. A small cyst in a mass of chronic mastitis usually feels very hard, but gives a sense of elasticity and is frequently accompanied by other patches of mastitis or cysts. The ill-defined limitation of this mass, the very firm, inelastic feel, and the apparent deep connection with the lactiferous ducts were all points in favour of its being carcinomatous, and the patient was advised to undergo operative treatment.

In discussing the treatment to be adopted Mr Swan strongly advocated the removal, not only of the breast and the axillary tissues, but also of both the sterno-costal portion of the pectoralis major and the whole of the pectoralis minor muscles, together with the aponeurosis covering the upper part of the rectus abdominis muscle. In this way the axillary lymphatic area can be removed as high as the clavicle and a free exposure is given of the small triangular space above the upper border of the pectoralis minor muscle, where small glands draining the deeper portions of the breast are present.

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Mr. Swan insisted on the great advantage of keeping the upper part of the primary incision well above the axillary fold, for he had shown that the ultimate movement of the arm was very much better where the scar after the operation did not encroach into the axilla—in fact, very free movement is ensured.

CASE 2. *Carcinoma of the rectum.*—Female, æt. 58 years, came up complaining of discomfort in the left iliac fossa. Except for occasional constipation she had noticed no trouble with the bowels until the previous week, when, for the first time, she found the motion streaked with some blood-stained mucus, which she attributed to straining at stool. She had experienced a feeling of weight in the left iliac region for some months and had been treated for flatulence. She had not lost weight.

The patient was a well-nourished, healthy-looking woman. No tumour could be felt in the abdomen; the gastric and colic resonance was normal. The left kidney could not be felt, nor was there any increased frequency of micturition. *Per vaginam* the uterus was small and no tumour was felt in the lateral fornices, but upon digital examination of the rectum the lower end of a growth could just be reached by the finger-tip. The growth occupied the posterior and lateral aspects of the rectal wall and seemed movable in the sacral concavity. No glands could be felt to be enlarged in the lateral pelvic space.

This case showed the frequent insidious nature of a rectal carcinoma, which may reach a comparatively advanced stage before any symptoms attract the patient's attention. Mr. Swan laid stress upon the value of a rectal examination in any suspicious case, if necessary under an anæsthetic, when the mobility of the tumour in the surrounding tissues can be more easily ascertained. In discussing the treatment of this case Mr. Swan advised the removal of the whole pelvic colon, rectum, and anal canal by a combined abdominal and perinæal operation, after a preliminary colostomy opening had been made. Although the operation was severe it had the following advantages:

(1) That the growth could be removed with a free length of bowel above and below it.

(2) That the posterior and lateral lymphatic areas extending from the growth could be removed with the pelvic cellular tissue.

(3) That hæmorrhage was entirely under control by ligature of the superior hæmorrhoidal artery, whilst important structures, such as the ureters, were under direct vision and avoided.

(4) That the patient was left with an efficient colostomy opening, instead of a sacral fistula which often resulted from a trans-sacral resection of the rectum.

(5) That a trans-sacral operation would be difficult in this particular case owing to the distance of the growth from the anal orifice.

(6) That the operation gave much more chance of ultimate cure than the trans-sacral excision owing to the more extensive removal of lymphatic areas, and that the cases in which it had been performed in the Cancer Hospital showed good results up to the present, although sufficient time had not elapsed to speak of them as cures.

CASE 3. *Epithelioma of the lip.*—Male, æt. 68 years, presented himself for a fungating tumour of the lower lip. This commenced as a "pimple" to the right of the middle line of the lower lip, about twelve months previously, and had gradually increased in size. Patient had been a confirmed smoker, carrying his pipe on the right side, but had not smoked a clay pipe for many years. Soon after the lump appeared the surface ulcerated and he was obliged to smoke his pipe on the left side, as it caused pain in the ulcer.

On examination, a fungating ulcer extended from the left side of the mid-line of the lower lip to within half an inch of the angle of the mouth on the right side. The outer free margin was prominent, with firm rugose edges, but the inner aspect was more infiltrated and extended in the mid-line by direct continuity on to the mucous membrane reflection on the anterior surface of the mandible in a small area the size of a threepenny piece. A few glands could be felt slightly enlarged in the right sub-maxillary area, and another small one at the situation of the bifurcation of the carotid vessels. The patient had no difficulty in mastication, but was troubled with slight dribbling of saliva.

Mr. Swan discussed the advisability of operation in this case, pointing out the chronicity in many cases of lip cancer, but particularly the terrible suffering of these patients when the growth is left untouched, when from the gradual spread of the disease the cheek becomes affected, the cervical

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glands break down and ulcerate through the skin with the consequent danger of hæmorrhage. Putting this before the patient, he advised him to undergo an operation, planned to remove the whole lower lip together with the anterior layer of the mandibular symphysis, at the same time removing the cervical, sub-mental, and sub-maxillary lymphatic glands, together with the salivary gland. The skin flaps could be brought up to cover the mandible and fashioned to make a new lower lip. Mr. Swan showed some specimens from the Museum of extensive lip cancer he had removed in even more advanced cases than the present from patients who still remained well and free from recurrence, one nineteen and the other twelve months after their operation, and in comfort.

CASE 4. *Nephrectomy and ureterectomy for tuberculous disease.*—Female, æt. 38 years, came up for examination. She was sent to Mr. Swan in August, 1907, for profuse hæmaturia and pain in the left lumbar region. She had been well until two months previously when she first experienced severe pain in the left lumbar region, causing sweating, vomiting and collapse, and followed in a few hours by profuse hæmaturia. The blood lasted some days, gradually subsiding but frequently recurring, sometimes after similar acute attacks, but sometimes with only slight lumbar aching. In some of the attacks accompanied by pain she had noticed long, tapering clots in the bloody urine. During the intervals of hæmaturia she sometimes noticed an aching in the left loin; at other times she was quite free from pain. On examination in the interval of an attack, the left kidney could be felt enlarged and rounded and was slightly tender to the touch. The right kidney could not be felt. The case was admitted as one of probable malignant growth of the kidney. After a few days' rest in bed and when the urine contained no blood a cystoscopic examination was made. The bladder was normal, but the left ureteric orifice was found retracted and drawn up so as to open at the apex of a funnel-shaped depression. No urine was seen to flow from it. The right orifice was normal and clear urine was emitted with vigorous efflux. A Luy's segregator was introduced and four ounces of clear urine (sp. gr. 1018, urea 1·8 per cent.) collected from the right side, but none flowed from the left side. Some few days later when hæmaturia was again present, the cystoscope showed the blood to issue from the left ureter, whilst clear urine passed from the right. The segregator was again used and after a short interval clear urine was obtained from the right side and deeply blood-stained fluid from the left side, the percentage of urea being 2·2 on the right and ·8 on the left. On vaginal examination on this occasion, the left ureter was felt to be distinctly thickened.

The left kidney was then explored and found enlarged with numerous softened tuberculous cavities; the pelvis was distended with blood-clot. The lumbar incision was lengthened downwards and forwards, and the peritoneum detached from the iliac fossa, the thickened ureter being exposed. The renal vessels were ligatured and divided, and the ureter then separated as far as could be reached under the broad ligament, ligatured with catgut and divided, the stump being scraped and painted with phenol. The wound was closed with a lumbar and inguinal drainage-tube. Except for a slight tuberculous infection of the upper part of the wound (which was subsequently excised), the patient did well and now comes up for treatment with tuberculous vaccine, by which her opsonic index is maintained about 1·3. She has put on weight, has no increased frequency of micturition and her urine is quite normal. The extensive scar of the operation is quite sound.

Mr. Swan pointed out the difficulties in diagnosis in this case where the prominent symptom was profuse hæmaturia. Doubtless the attacks of renal colic were due to the passage of a blood-clot down the ureter, but a true diagnosis should have been made when the ureter was felt thickened *per vaginam*. Unfortunately the urine was not examined for tubercle bacilli. Tuberculous nephritis is frequently accompanied by slight hæmaturia, but more often by pyuria and frequency of micturition. No pus was ever detected in this case, probably from the fact that the ureter on the affected side was blocked by blood-clot, and that the urine that was collected was that excreted from the healthy right kidney. Although a small portion of infected lower end of the ureter was left in this case it caused no symptoms, and in several similar cases the patients have had no subsequent trouble from it, even after the lapse of three years.

CASE 5. *Carcinoma of the anterior wall of the stomach; partial gastrectomy.*—D. R—, æt. 52 years, a cook, was admitted under Mr. Swan in August, 1906, for abdominal tumour. She had had complete absence of subjective symptoms, and had only noticed the tumour for about a fortnight. She had lost weight for some months. On examination, a mass could be felt in the abdomen to the left of the umbilicus about the size of a closed fist.

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It caused no pain, but examination was difficult owing to muscular rigidity. Under an anæsthetic the mass was found to be movable in the abdominal cavity; it could be pushed up under the costal margin, laterally to the right of the umbilicus and downwards towards the iliac fossa. No pedicle could be traced from it into the pelvis and the left kidney could be felt in the normal position. The mass was slightly irregular, gave no sense of fluid, and no other tumour could be felt in the abdomen. The abdomen was opened by an incision through the left rectus muscle and the mass was found to be a large carcinoma commencing in the anterior wall of the stomach and fungating into the lumen of the latter. (The specimen was brought from the Museum.) The whole stomach was elongated, and with no adhesion to any other organ allowed of the free mobility of the tumour. A few small glands were found immediately along the greater curvature of the stomach, but none could be felt in the portal fissure, and the liver was normal. The whole mass was easily delivered from the wound and the abdominal cavity packed off with towels. An incision was next made into the healthy wall of the stomach on the proximal side of the tumour, and the interior explored by the finger. The tumour fungated into the lumen of the stomach in a cauliflower form, but with no actual ulceration. The opening was temporarily closed and clamps applied on each side wide of the growth, including between them a large piece, somewhat wedge-shaped, of the stomach, after the lesser sac had been opened through the gastro-colic omentum. The corresponding sections of gastro-hepatic and gastro-colic omenta were ligatured as wide of the stomach as possible and the large piece of stomach resected. It was then found possible to bring the proximal and distal segments together with practically no tension, and so having sewn up the upper portion of the opening in the proximal segment, the two openings were joined by direct anastomosis with three layers of sewing thread, the gastric colic omentum being grafted on to the lower portion of the junction. With the absence of tension on the sutures a second gastro-enterostomy opening was not deemed necessary. The patient did very well and left the hospital, taking a full solid diet in five weeks.

You can see her yourselves to-day in robust health. She has put on over two stone in weight, can take any food without discomfort, and no sign of recurrence can be found on examination. The liver is not felt, nor can any abdominal mass be felt. This is the piece of stomach resected, and by the opening subsequently made in the posterior aspect you can see the large mass of growth fungating from the anterior gastric wall. Microscopically the growth proved to be carcinoma.

Mr. Swan commented on the unusual features of this case, pointing out that gastric carcinoma was usually of the infiltrating or ulcerating type with tendency to invade other organs, and mentioned recent cases where he had found direct infiltration into the left lobe of the liver and into the pancreas. Also the free mobility of this mass from the gastric elongation and the complete absence of vomiting or gastric symptoms.

CASE 6. *Hour-glass contraction of the stomach.*—G. C—, male, æt. 42 years, came up to report his progress. He was admitted under Mr. Swan in October, 1905, for severe vomiting and rapid emaciation. Vomiting commenced about three months previously, but for the last five weeks had been much worse, any form of fluid being almost immediately vomited. He had lost flesh rapidly, but had never had hæmatemesis. He had been under treatment, but discharged from a general hospital as an inoperable case of pyloric cancer.

On examination he was terribly emaciated, weighing 6 stone 8 pounds. No tumour could be felt in the abdomen, but as he seemed to be rapidly starving Mr. Swan opened his abdomen to perform gastro-jejunostomy. The stomach was found to be a very marked example of hour-glass contraction, a very marked constriction separating the larger proximal portion from the smaller distal. An opening was made in the proximal portion and the finger introduced, when it was found that the cicatrisation was so marked that the tip of the finger was tightly gripped in the opening between the two portions of the stomach. This exploratory incision in the stomach was sewn up and a long horizontal incision carried through the anterior wall of the stomach, dividing the constriction. The median portions of this incision were drawn apart and the incision sewn up vertically, thus obliterating the opening, some difficulty being found in the suturing owing to the great muscular hypertrophy at the proximal side of the obstruction. The patient's condition was too bad to allow of a gastro-jejunostomy being performed in addition and the wound was sewn up. The patient made an excellent recovery and you see him now in good health. His weight is 9 stone 4 pounds, and until the last month he has been able to take any form of food. Recently he has had some slight indigestion after a full meal, and Mr. Swan suggested that cicatrisation may have again produced some stenosis, and suggested the possibility of having to perform a gastro-jejunostomy at a later date.

Mr. Swan pointed out the difficulties in the diagnosis of gastric cancer, and suggested that an earlier exploratory operation in these cases would often prove that a radical operation might be performed, or at all events a palliative short-circuiting anastomosis. There was no history of corrosive poisoning in this case.

*September 14th*, 1908.

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## ATONIC DILATATION OF THE STOMACH.

By ERNEST YOUNG, M.D., Dunelm.

I PROPOSE considering atonic dilatation of the stomach, discussing briefly its ætiology, symptomatology, diagnosis, and prognosis, describing in greater detail the various methods of treatment.

Atonic dilatation was first recognised towards the end of the eighteenth century by Johann Peter Frank, who separated it sharply from the forms caused by stenosis.

Of late years, however, considerable confusion has arisen with regard to the exact nature of dilatation, chiefly owing to the erratic nomenclature applied to the condition, various authors substituting terms such as "mechanical insufficiency," "myasthenia gastrica," "ischiochymia," etc. It may be well, therefore, before proceeding further to endeavour to obtain a clear conception of what the term "atonic dilatation" really indicates. Putting aside secondary or obstructive dilatation as outside the limits of my subject, and taking for granted that atonic dilatation is recognised as primary or non-obstructive in origin, it may be said to embrace three factors—(1) atony, (2) enlargement, (3) undue retention of food. Lacking one or more of these three factors it cannot fairly be called atonic dilatation.

From the above data, then, the condition may be defined as "a primary or non-obstructive dilatation due to atony of the stomach wall and resulting in undue retention of food."

For clinical purposes it is convenient to divide the condition into two degrees, according to the duration of retention of food. Thus we have to consider:

(1) *Dilatation with partial retention*, that is, when the stomach cannot expel its contents within six or seven hours after a meal, but is able to do so during the long interval of the night.

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(2) *Dilatation with complete retention*, that is, where the stomach cannot empty itself even during the long interval of the night and therefore always contains some residua of food.

By far the greater number of cases of atonic dilatation correspond to the first degree, comparatively few entering the second degree, this more serious condition being generally due to obstruction. Saundby (1) and others even go so far as to say that "atonic dilatation of the stomach never causes complete retention of food," but this is contrary to the experience of Broadbent (2) and other observers. Personally I have seen three such cases, two of which may be quoted:

(1) Mr. E. S—, æt. 20 years. Rheumatic fever at sixteen; dyspeptic symptoms since convalescence. Pain and oppression in upper abdomen after food. Anorexia, constipation, great emaciation, no vomiting.

*Objective.*—Lower border of stomach extends nearly to pubes, upper border normal, no signs of obstruction. Food residues in stomach before breakfast.

*Diagnosis*, atonic dilatation. Complete cure in fifteen months by careful dietary, lavage, and faradism with intra-gastric electrode.

(2) Miss M. V—, æt. 29 years. Dyspeptic symptoms for last ten years; been treated for nervous dyspepsia and gastric catarrh. Most prominent present symptoms are, "dragging pain" in epigastrium, flatulence and constipation, vomiting about once a fortnight; is very weak, and has lost a great deal of weight (from 8 st. 4 lb. to 6 st. 8 lb.) for the last year.

*Objective.*—Splashing sound obtainable several hours after food, extending three fingers' breadths below the umbilicus. "Food residues in stomach before breakfast"; no gastroptosis, no signs of obstruction.

*Diagnosis*, atonic dilatation. Cured in nine months by careful dieting and lavage.

So far we have discussed only the ordinary or chronic form of atonic dilatation, with which we are chiefly concerned. The rare condition of acute dilatation, will be briefly discussed at the end of ætiology.

## Ætiology.

According to Saundby (3), atonic dilatation is most frequent between the ages of twenty and fifty; occurs twice as often in women as in men, and is ten times more common than obstructive dilatation.

The chief factors which tend directly or indirectly to produce the condition are:

(1) *Heredity.*—Heredity undoubtedly plays a more important part than is generally attributed to it, and Boardman Reed (4) goes so far as to say "that the children of parents so afflicted do not often escape it."

While it is undeniable that "weak stomachs" run in some families, probably many of the so-called hereditary cases are produced by improper feeding in infancy or childhood.

(2) *General mal-nutrition.*—Another cause is general mal-nutrition and debility, such as may arise during convalescence from acute infectious fevers, where the stomach, sharing in the general bodily weakness and probably poisoned by toxins from the fever, is especially prone to dilate. A single indiscreet meal at such a time may give rise to a troublesome and more or less permanent dilatation, while occasionally an acute condition may supervene. The writer has had a case in which the stomach was temporarily dilated for some weeks after influenza, the condition recurring again after a subsequent attack of influenza a year later.

Similar but less powerful prediposing causes are debilitating diseases such as chlorosis, phthisis, cardiac or renal diseases.

(3) *Neurasthenia.*—Saundby (5), and more recently MacCullum (6) have asserted that neurasthenia is the chief cause of the condition.

On the other hand Riegel (7) asserts, "It has not yet been proved that chronic ectasis may develop upon a purely nervous basis." The writer inclines to the latter opinion as, although some cases in which neurasthenia and dilatation are associated may possibly have a common origin, yet so far as cause and effect pure and simple are concerned, he cannot but think neurasthenia is, probably, far more often the result than the cause of atonic dilatation, owing to the general mal-nutrition and auto-intoxication caused by the latter.

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Whichever view is held, however, the neurasthenia should not be treated at the expense of the stomach, since the unlimited diet which is often prescribed in neurasthenia unaccompanied by dilatation cannot but have disastrous effects when dilatation is present, as the following will illustrate.

Miss M. L—, æt. 25 years. Had suffered more or less for five years from atonic dyspepsia associated with neurasthenic symptoms. Has had two six weeks' courses of Weir-Mitchell treatment within the last fifteen months, finishing the second course four weeks ago.

*Objective.*—Splashing obtainable almost to pubes at any hour of the day, food residua in stomach before breakfast; no signs of obstruction.

(4) *Gastric catarrh.*—Other diseases of the stomach, such as gastric catarrh, either in the acute or chronic form, may prove a powerful predisposing factor by reducing the value of the gastric secretion and promoting fermentation.

Hyperchlorhydria and hypersecretion are also said to be causative factors, but inasmuch as the dilatation produced is generally due to spasm of the pylorus, induced by a protective reflex from the duodenum (8), it must be regarded as more or less secondary in character.

(5) *Errors in diet.*—Errors in diet are a most fertile cause of atonic dilatation, whether the diet is excessive in quantity, poor in quality, or both combined.

Thus the condition may be induced in those who gourmandise, eat very rapidly, live on large quantities of coarse bulky food, or ingest excessive quantities of fluid such as beer, aërated water, or tea, the habitual over-distension of the stomach gradually producing atony of the walls of the viscus. Apart from excessive quantity, the condition may also be induced by normal amounts of food of insufficient quality, the stomach tending to share in the resulting mal-nutrition.

Many cases of atonic dilatation are also caused, undoubtedly, by faulty dietetic treatment of dyspeptic conditions, chiefly by prohibiting proteid food and ordering a "light" diet, consisting chiefly of carbohydrates.

As Professor Allbutt truly says (9), "There is no superstition more tenacious of life than that which prescribes carbohydrates to all dyspeptics as 'so digestible,' and into weak stomachs ready to dilate is thrown a mass of such a dish as rice pudding—a bulky food, imperfectly salivated and peculiarly apt to decompose with the disengagement of volumes of carbonic acid gas."

From cases he has seen the writer is quite convinced that in this way simple gastric atony is often converted into the more serious condition of atonic dilatation. One such case may be quoted:

Mr. E. C—, æt. 21 years. Had suffered from various dyspeptic symptoms for four years, consulting various doctors; was told he had atonic dyspepsia. Nearly all prescribed milk and milk foods, prohibiting or reducing to a minimum all meat foods. Dull heavy pain in epigastrium, flatulence, constipation, great emaciation (from 10 st. to 6 st. 4 lb.). No vomiting.

*Objective.*—Lower curvature of stomach midway between umbilicus and pubes; splashing seven hours after a meal; food residues before breakfast, but not constant.

*Diagnosis.*—Atonic dilatation. Cured in twelve months by diet, occasional lavage and faradism.

(6) *Gastroptosis* is often associated with atonic dilatation and is generally considered to be a predisposing factor. This, however, is denied by Loening (10) who claims that gastroptosis *per se* causes either no alteration of motility or an increase.

The dilatations resulting from nephroptosis are classed as "atonic" by Glenard, Debove, and Remond. That this condition may give rise to dilatation is denied by some writers, but that it may prove a direct causative factor is undeniable, since once the nephroptosis is relieved the dilatation is usually rapidly cured.

Miss K. E—, æt. 32 years. Suffered from dyspeptic symptoms, sensation of dragging in abdomen and pain in back for last three years.

*Objective.*—Lower curvature of stomach one inch below umbilicus. Splashing and food residua seven hours after a meal. No food residua before breakfast. Right kidney freely movable throughout a large area.

*Diagnosis.*—Atonic dilatation of first degree, nephroptosis of right kidney.

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*Treatment.*—Kidney supported in normal position by Ernst truss, careful dieting, triphase sinusoidal electricity, strychnine. Completely cured in three months.

Among other causative factors which have been described by various authors are—trauma in the gastric region (11), narcotics, especially alcohol and tobacco, organic lesions of nervous origin, idiopathic (12).

### Acute Dilatation.

Occasionally an acute type of dilatation appears, as already mentioned. Most frequently it arises in connection with acute infectious diseases, such as rheumatic fever, typhoid, pneumonia (13), influenza (14), etc. It may also supervene after a debauch, as a result of bulimia in diabetes, or after taking very cold food, such as an ice, on an empty stomach, as in a case quoted by Sir William Broadbent (15). Several cases have also occurred as a sequel to abdominal operations, the latest being reported by MM. Rautier and Walther (16). Cases have also occurred after chloroform narcosis preceding various operations (17).

The symptoms are sudden in onset and consist of marked painful swelling of the abdomen, nausea, vomiting, thirst, dyspnœa, and tendency to collapse. Partial or complete anuria may be also present.

A few cases of a very severe type, arising without obvious cause, have been reported by Fagge (18), Broadbent (19), Friedenwald (20), and others. In these cases—which Allbutt aptly describes as "overwhelming"—the stomach becomes enormously distended with fluid, apparently due to hypersecretion, the fluid tending to form again when evacuated.

The condition is characterised by more pronounced symptoms than the milder type, the vomiting being violent and intractable and accompanied by intense thirst, anuria, and finally collapse, death quickly ensuing in the majority of cases.

As to the cause of acute dilatation, some writers have thought the condition due to sudden paralysis of the gastric muscles, others to a spasm of the pylorus as a result of hyperacidity (21).

Treatment consists in lavage of the stomach until vomiting ceases, and rectal feeding until acute symptoms have subsided.

Returning to the subject of chronic dilatation its symptomatology may next be considered.

### Symptomatology.

The symptoms vary in more or less direct ratio to the duration of retention of food. The first symptom which generally attracts attention is a sensation of weight, flatulence, and discomfort in the upper abdomen after food, occurring at first only after a large meal, but gradually tending to appear whenever food is taken. The flatulence may be associated with pyrosis and gaseous eructations, either tasteless or flavoured with food.

There is little actual pain at first other than perhaps a dull interscapular aching.

The above is fairly typical of the early stages of the condition, but as the stomach becomes more dilated, the retention of food longer, and abnormal fermentation more pronounced, other and more serious features become apparent. Thus, a dry, harsh skin, thirst, scantiness of urine, and constipation appear, owing to the dehydration of tissues which occurs, partly as a result of hypersecretion, partly from the small amount of fluid which the stomach is able to pass into the duodenum.

The urine is scanty, high coloured, often contains triple phosphates, and is usually deficient in chlorides.

It may also be alkaline and generally shows marked indicanuria as the result of intestinal fermentation.

If gastritis is not present *ab initio* the abnormal fermentation in the stomach and consequent formation of irritant organic acids generally gives rise to it, and may determine similar intestinal trouble, mucous colitis being a fairly frequent complication.

Emaciation and loss of strength become progressively marked owing to the dehydration of the tissues, and to the fact that whatever food does enter, the intestine has been so disorganised by abnormal gastric fermentation as to have but little nutritive value (22).

The appetite is not affected much at first, but anorexia soon becomes apparent, although in a few cases bulimia has been noticed.

Vomiting in atonic dilatation, as Maynard has pointed out (23), is distinctly uncommon. In my experience it occurs only in the comparatively rare cases of complete retention, and even then is of a

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milder type than in obstructive dilatation, although otherwise characterised by the same features.

It may occur daily, every two or three days, or very irregularly. The attack generally takes place towards the end of the day or at night, only rarely in close proximity to meals. It is seldom accompanied by retching or nausea, although according to Riegel (24) it may occasionally be preceded by cramp-like sensations, or even violent pains.

The vomit is usually greyish in colour, with intensely sour smell, owing to the organic acids present. On standing it divides into the usual three characteristic layers, the lowest consisting of food, the middle of decidedly acid fluid, the upper of brownish froth containing various bacteria and fungi, chiefly yeast and sarcinæ.

Various symptoms, more or less due to auto-intoxication, may be present, such as headache, depression, insomnia, irritability and inability for physical or mental exertion.

Urticaria and erythema have been noted and described as toxæmic in origin, although possibly they may be produced by reflex irritation transmitted to cutaneous nerves. Vertigo may also arise, being caused, according to Sir William Broadbent (25), entirely by flatulence and consequent mechanical pressure, since it is quickly relieved by the eructation of a few c.c. of gas. Other possible symptoms mentioned by him are irregular heart and pulse and anginoid pain, apparently produced by actual pressure upon and embarrassment of the heart.

Tetany is a peculiarly interesting but rare symptom which, according to Hemmeter (26), is seen most frequently in those cases associated with hypersecretion. The condition, which is usually preceded by exhaustive vomiting, or may even be initiated by lavage, is characterised by contraction of the fingers or toes, or both, sometimes the arms and legs being also involved, and more rarely the muscles of the trunk, neck and face. Clonic convulsions may alternate with the true seizures.

How tetany is produced is still undecided. Some, with Kussmaul, attribute it to the abnormal dryness of the tissues, this view having been championed of late years by Fleiner (27).

Another theory propounded by Germain Sée is that the spasms are due to the irritated nerves of the gastric mucous membrane causing a reflex stimulation of the central and peripheral nervous systems.

The third is the toxæmic theory, which is, that the condition is due to the absorption of poisons formed in the stagnating fermenting contents of the dilated stomach. From experiments performed by Halliburton and McKendrick in 1901 most clinicians incline to this view.

Another interesting symptom not mentioned in the usual text-books is the phenomenon described by Bouchard (28), viz. nodosities on the phalango-phalangeal joints of the fingers, formed by the enlargement of the bases of the second phalanges. The writer has noticed this feature in two cases of atonic dilatation.

## Diagnosis.

To establish a diagnosis of atonic dilatation of the stomach we must—

(1) Demonstrate its enlargement.

(2) Demonstrate its atony or lack of motor power.

(3) Demonstrate the absence of obstruction.

To demonstrate its enlargement the following are a few of the methods which may be employed.

(*a*) Inspection, palpation, percussion and auscultatory percussion, either with or without artificial inflation of the stomach.

(*b*) By the introduction of a sound and palpating same through the abdominal wall.

(*c*) By the electro-diaphane.

(*d*) By the X rays.

To ensure accuracy it is generally advantageous as a preliminary measure, provided there is no suspicion of ulceration or advanced malignant disease, to inflate the viscus either with carbonic acid gas, or with air pumped through a stomach-tube, or by Spivak's (29) ingenious auto-inflation tube.

The first method is the most convenient, much less disagreeable to the patient, and quite as efficacious for clinical purposes. That it is often unsatisfactory is due to the fact that the gas is not generated in sufficient quantities, the quantities of sodii bicarb. and ac. tartaric which are generally given being far too small. The best doses to give are those advised by Saundby (30) viz. 120 grains of bicarbonate of soda dissolved in half a tumbler of water, preceded or followed by ninety grains of tartaric acid similarly dissolved. It is better, however, always to give the acid solution first, so that the alkaline solution which follows may neutralise any acid adhering to the teeth.

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Saundby claims, and truly, that these large doses do not cause pain or undue discomfort and efficiently demonstrate the size of the stomach, which the smaller powders may fail to do.

Having in this manner efficiently distended the stomach one can often detect enlargement by inspection alone if the abdominal walls are fairly thin. Thus, if the greater curvature of the stomach can be detected below the umbilicus the viscus is dilated, provided the upper curvature is in its normal position, that is, hidden beneath the liver and the margin of the ribs.

If, however, there is a hollow in the epigastrium beneath which the upper curvature of the stomach may be detected, the condition is one of gastroptosis, which is not necessarily accompanied by dilatation.

Gastroptosis is more easily determined with the patient in the erect position, and, even for this fact alone, it is always advisable to examine patients both in the erect and recumbent positions.

By palpation it is possible at times to feel the lower margin of the stomach, but its chief use is for obtaining a splash by succussion or clapotage at an abnormal time. Thus, although a slight splash may be considered normal to within two or three hours after a meal, if it can be produced six or seven hours after a meal it indicates defective motor power, and if it can be elicited over a larger area than that normally occupied by the viscus it also denotes dilatation.

Percussion and auscultatory percussion are more generally useful than either inspection or palpation, since they enable us to determine the upper limits of the fundus. This is an important point in differential diagnosis, since the enlargement of the fundus—that is, the upward dilatation—is usually greater in atonic than obstructive dilatation (31), occasionally reaching as high as the fourth rib.

The lower limit of the stomach is sometimes difficult to define if the stomach has not been well inflated, since its percussion note is very similar to that of the colon if the latter is flatulent. Should there, however, be any doubt in the matter, it is well to make the patient drink one or two glasses of water and examine in the erect position, when the percussion note of the stomach, formerly tympanitic all over, will now be dull in the region of its lower curvature, and may thus be differentiated from the tympanitic note still present in the colon.

Another method of differentiation was pointed out by Broadbent (32), who showed that the stomach and colon might be differentiated by means of auscultation, the cardiac sounds, especially the second, being heard all over the area of resonance proper to the stomach with a characteristic tympanitic consonance, which is lost the moment the stethoscope passes from the stomach to the colon.

The above means, namely, inspection, palpation, percussion and auscultatory percussion, are generally sufficient to establish a diagnosis of dilatation. If further proof is required, an intra-gastric electrode, such as Boas's (a metallic spiral sound enclosed in a stomach-tube), may be introduced into the stomach and palpated through the abdominal wall. The sound is readily palpable as a rule, and the writer has found it an excellent means of mapping out the greater curvature.

The electro-diaphane and X rays are seldom indispensable, although the latter, since the introduction of the orthodiagraph and other improvements in technique, is invaluable in differential diagnosis, and will be discussed further under that heading.

To determine atony or weakened motor power of the stomach is a comparatively easy matter. Thus, if a splashing sound can be obtained seven hours after a test dinner, and if by the subsequent introduction of a stomach-tube food residues are withdrawn, then motor insufficiency, at any rate of the first degree, is established. If, further, splashing and food residues from an evening meal can be obtained before breakfast, then motor insufficiency of the second degree is established.

Other tests, such as Ewald's salol test, Sahli's desmoid test, Fleischer's iodoform test, or the iodipin test may be substituted for the above if for any reason it is undesirable to pass a tube. They are, however, not nearly so satisfactory and trustworthy as the test meal.

Hemmeter (33) has devised an apparatus for testing gastric peristalsis which deserves a passing reference. It is called the "Gastrograph," and consists of an intra-gastric stomach-shaped rubber bag connected by means of a tube with a manometer, kymograph and tambour. When the bag is introduced into the stomach and inflated, the periodicity and also the amplitude of the peristaltic movements are recorded on the tambour. Hemmeter claims

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that most exact results are obtained, but while admiring the ingenuity of the method one can but endorse his own opinion concerning it—"probably none of these apparatuses will be regularly used in practice."

Having determined the enlargement and atony of the stomach, we have now to exclude obstruction, a process that entails differential diagnosis between obstructive and non-obstructive dilatation.

In differential diagnosis we can learn much from examination of the stomach contents. Thus, coarse, undigested remains of food, particularly of meat, absence of free hydrochloric acid, abundance of lactic acid, perhaps Oppler-Boas bacilli and altered blood, primarily indicate carcinoma. On the other hand, a profuse residue with remains of starch and the presence of free hydrochloric acid, yeast, and sarcinæ would favour benign dilatation.

In benign obstruction a thickening may possibly be felt in the region of the pylorus; visible peristalsis is almost invariably present, while, as already noted, the upward distension of the viscus is less marked and the downward distension is usually more marked than in atonic dilatation.

In malignant obstruction a tumour may or may not be felt, but a careful consideration of the history and the character of the vomit will often clinch the diagnosis, although as Hale White (34) and others have shown, malignant obstruction of the pylorus is sometimes accompanied by normal gastric chemistry. I myself had such a case last year:

Mr. E. B—, æt. 54 years. Symptoms of three months' duration. Gradually increasing anorexia; loss of strength and emaciation; no particular pain; vomited twice.

*Objective.*—Greater curvature of stomach three inches below umbilicus, lesser curvature normal. Slight thickening detected at pylorus. Gastric chemistry normal, plenty of free hydrochloric acid; no lactic acid, no Oppler-Boas bacilli.

*Diagnosis.*—Previous to gastric analysis suspected malignant obstruction; after analysis diagnosed benign obstruction, the surgeon concurring. At operation, however, a scirrhous mass was found encircling the pylorus.

As already mentioned the X rays may be of the greatest value in differentiating between atonic and obstructive dilatation, thanks to the pioneer work of Cannon in America (35), and its extension by Rieder in Munich and Holznecht in Vienna. Hertz (36), in a paper to the 'Practitioner' for April, 1907, briefly describes the technique, which is as follows:

Half a pint of bread and milk containing about an ounce of bismuth sub-nitrate is given on an empty stomach and the patient examined at regular intervals of half an hour or an hour. When great precision is desired orthodiagraphy may be employed. Not only may the exact size and position of the stomach be accurately determined, but a diagnosis between atonic dilatation and pyloric obstruction can easily be made by noting the frequency of the peristaltic contractions. Normally, three waves pass to the pylorus every minute, while in atonic dilatation the waves are less frequent, and in obstruction more frequent than normal.

The differential diagnosis of gastroptosis from atonic dilatation has already been mentioned.

The condition to which Riegel has given the name "megalogastria" will hardly require to be differentiated from atonic dilatation, since it seldom, if ever, gives rise to symptoms, being usually discovered by accident.

In this condition, which occasionally occurs in gross feeders and those who live on bulky vegetable foods, the stomach may greatly exceed the average normal limits, but yet is capable of expelling its contents into the duodenum at the proper time, and so, clinically, cannot be regarded as dilated.

### Prognosis.

The prognosis depends chiefly upon the underlying cause of the dilatation. Thus, if atonic dilatation occurs in relation to an acute infectious disease, it generally yields more readily to treatment than when associated with more chronic conditions such as phthisis, cardiac or renal disease, improvement of which must necessarily occur before the dilatation can be materially benefited.

Turning, however, to the more frequent cases, viz. those which are purely dyspeptic in origin, we may speak more dogmatically. In such cases, if only partial retention of food is present, the majority may be cured by careful treatment. Even in cases of complete retention a hopeful issue may be anticipated provided the condition

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is of fairly recent origin. If it is of longer standing and associated with severe gastritis, fibrous changes may have developed in the musculature of the stomach wall and so a complete cure is impossible.

The condition is, however, far from hopeless, as by careful dietetic and electrical treatment the viscus may be considerably reduced in size, and although it cannot be brought back to the normal condition, yet by careful dieting and occasional lavage the patient is able to lead a very tolerable life.

Although, as stated, many cases can be cured, the condition is, nevertheless, one which is very liable to recur on the advent of any great mental or physical strain or enforced change in dietetic habits, and this is an important point to remember with regard to life insurance.

A feature which I have noticed and which may possibly be of clinical interest is the fact that patients who have suffered from atonic dilatation always tend to retain an epigastric tenderness.

## Treatment.

The essential factors for successful treatment in atonic dilatation of the stomach are:

(1) To strengthen the musculature of the viscus and promote peristalsis.

(2) To arrest fermentation and thereby inhibit the symptoms of auto-intoxication.

In endeavouring to attain these objects the primary consideration must be to regulate the diet, and unless this is done we cannot hope for a satisfactory issue, despite the aid of other therapeutic measures which, although valuable, can only be regarded as accessories. Briefly, the ideal diet is one which combines "a maximum of nourishment with a minimum of weight and bulk," and should, therefore, consist chiefly of proteid foods in an easily assimilable form, such as minced freshly-cooked beef, mutton, chicken, game, fish, and the various meat and milk powders.

Starchy food, in view of its weight, bulk and fermentability, should be reduced to a minimum, and in severe cases may occasionally be excluded altogether during the early stages of treatment. When given, it should be in the form of crisp toast, rusk, or "baked bread," (thin slices of bread baked in the oven until golden brown), in all of which the starch is partially dextrinised.

Vegetables, for a similar reason as regards bulk and fermentability, should generally be excluded in the early stages of treatment, and when introduced should be of green varieties only, and given in the form of purées.

Fats should also be but sparingly partaken of, cream or fresh butter being the most digestible forms when allowed.

In order, again, to minimise weight and bulk no fluids should be taken with meals, but from five to ten ounces of water, preferably hot, may be slowly sipped one hour before meals. Taken in this way, as Sir William Broadbent (37) has pointed out, "it stimulates the stomach to contract and expel gas or stagnant contents." In fact he considers in common with Allchin (38) and others, that in severe cases a diet restricted for a time to lean minced beef and hot water may be most beneficial. Saundby (39) says, "the digestibility of finely divided meat is not sufficiently appreciated; probably no food is tolerated so well by our stomachs, for even where the gastric chemistry is deficient such food is easily got rid of and causes no discomfort." The writer, from personal experience and from results obtained with patients, can emphatically endorse this opinion. The rationale of the method, sometimes called "the Salisbury diet," is as follows: The patient is restricted to three meals a day, with a five hours' interval between each. The diet for a few weeks is restricted to minced freshly-cooked beef, varying from two to eight or ten ounces at each meal, and hot water, the latter being slowly sipped in quantities of five to ten ounces one clear hour before each meal. Ten ounces should be the limit, since the bulk of a pint, which is sometimes prescribed, is obviously injurious to a dilated stomach. As soon as evidence of undue fermentation has disappeared, a little starch food is added to the dietary, then purées of green vegetables, and, as progress is made, various other articles of diet at the physician's discretion.

Summing up the advantages of the above method it is apparent (1) that the small bulk and comminuted condition of the food imposes but a minimum of work on the stomach; (2) the hot water tends to stimulate and cleanse it; (3) the absence of carbohydrate food, by depriving abnormal fermentative organisms of pabulum, tends to arrest the symptoms of auto-intoxication.

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In milder cases small quantities of starch and fat foods may often be taken with safety from the commencement of treatment, but even in these cases the proteid element should predominate in the dietary until the dilatation has markedly decreased.

In very severe cases it is often advisable to confine patients to bed and feed exclusively by nutrient enemata for some days, so giving the stomach a complete rest. Even when food by the mouth is resumed, it is well, until the patient is able to take a fair amount of nourishment, to continue them, remembering also to give enemata of water up to two pints a day, since, in such a case, the patient cannot with safety take enough fluid by the mouth to suffice for his physiological needs.

In these severe cases the diet should be increased only with the greatest caution, and it is well to have some clinical method to decide when and how it may be altered with safety. Various German and American authorities recommend periodical test meals with subsequent analyses, determining from the appearance of the food residues and the proportion of acids and ferments present what articles of food should and should not be taken. This method at best is only diagnostic of digestion in the stomach, ignoring altogether the far more important final digestion which takes place in the intestine, and so the patient may possibly be deprived of some article of food which, although not thoroughly digested in the stomach, might, nevertheless, have been adequately dealt with in the intestine. In the opinion of the writer a far more accurate judgment may be formed by examination of food residua upon which all the digestive juices have acted, namely, the fæces. Thus if, say, from the fæces of a patient on a diet of meat and starch food we find, chemically, an intensely acid reaction, and microscopically, an enormous amount of abnormal fermentation (evidenced by abundance of bacteria, fungi, liquefied mucus and gas bubbles) combined with a quantity of only partially digested starch, and perhaps some unaltered starch granules, while the remnants of meat present are well broken up, are more or less homogeneous in appearance and show no marked muscle striation, we may conclude that although the meat is being well digested the starch is not, but is causing abnormal fermentation with the production of organic acids, the inference naturally being to eliminate starch food from the dietary until the abnormal fermentation has disappeared. The above only touches the fringe of coprology, but I claim that the results which may be obtained are far more helpful than those of gastric analysis, and the patient escapes the unpleasantness which a test meal and its subsequent extraction entails.

*Drugs.*—In drugs, as in diet, the twofold object must be to promote peristalsis and counteract fermentation. Probably the drug *par excellence* in promoting peristalsis is strychnine, which, according to Hemmeter (40) should be given in large doses, not less than gr. $\frac{1}{24}$, *t.i.d.* Suprarenal extract, ergot, creasote, and orexin are a few of the various other drugs which have been given with the same object, but it is doubtful if any are as valuable as strychnine.

With regard to anti-fermentatives, it must be confessed that although frequently efficacious in the milder forms of dilatation the results are often disappointing in the more serious cases, seeming, apparently, to show that even the strongest antiseptics we can administer compatible with safety will only control a very limited amount of fermentation. Amongst anti-fermentative drugs which various authorities have claimed to be useful may be mentioned calomel, salol, sulpho-carbolate of soda, salicylic acid, salicylate of bismuth, carbolic acid, creasote, resorcin, and ammonium and sodium benzoate. From experiments made by Kuhn on several anti-fermentatives he found salicylic acid one of the most useful. Hydrochloric acid is also popular with many, but should be given with care if much gastritis is present. Armstrong (41) recommends from 6–12 minims of fuming hydrochloric acid in six ounces of water shortly after meals. This is often very beneficial, as apart from its antiseptic properties it remedies the deficiency of hydrochloric acid which is frequently present in the gastric secretion. In endeavouring to thus reinforce the gastric secretion, it is said to be doubtful whether it is ever necessary to give pepsin. Riegel (42) points out that, "Whenever we find free hydrochloric acid in the stomach there is also a sufficiency of pepsin, and, even if hydrochloric acid is absent, pepsin is present, or at any rate its precursor pepsinogen," which only requires a dose of hydrochloric acid to call forth its activity. In

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contra-distinction to this, however, it cannot be denied that the administration of hydrochloric acid may occasionally cause only increased pain and discomfort, while pepsin given alone will often greatly improve the condition.

In connection with gastric antisepsis magnesium peroxide must also be mentioned, since it possesses decided value in this direction owing to its power of evolving nascent oxygen when decomposed by the gastric juice.

I have seen marked benefit in two cases from a combination of peroxide of magnesium and poplar charcoal made up in the form of biscuits.

*Lavage.*—Lavage in atonic dilatation is a two-edged sword. Although a powerful therapeutic weapon in suitable cases, its indiscriminate use, particularly when combined with faulty technique, such as pouring three or four pints of fluid into a stomach before removal, has undoubtedly tended in many cases to aggravate instead of help the condition.

What, then, are the cases in which lavage should be employed? In cases of partial retention, unassociated with severe gastritis, it is seldom necessary once the diet has been carefully adjusted, but in the rarer cases of complete retention, where food residues are found in the stomach before breakfast, it should always be used until improvement takes place. The best time for lavage is a disputed question. Some authorities say "at bedtime," so as to ensure several hours' complete rest to the stomach. Others say "before breakfast," so that the stomach may absorb all the nutriment it possibly can during the night. Personally, my own opinion is that lavage before the evening meal, as advocated by Riegel (43), is the best method. By this means the "spoiled stuff" left from the two earlier meals of the day is removed, and the evening meal descends into a clean stomach, which almost invariably disposes of it satisfactorily during the night, although, lacking the lavage, food residues would have been present in the morning.

As to apparatus, I prefer a stomach-tube fitted with a rubber bulb. By means of the bulb the fluid injected "swirls" around the stomach with some force and so removes mucus far more readily than when the fluid is simply poured in by means of a funnel. An additional advantage is that aspiration may be performed if the fluid does not return readily, as is fairly often the case in atonic dilatation.

Many solutions are recommended for lavage, such as boric acid, salicylic acid, tannic acid, resorcin, etc., but plain warm water is probably the best and safest, although one drachm of bicarbonate of soda to the pint of water is useful if much mucus is present. Not more than three quarters of a pint should be introduced at a time, and it is generally sufficient to repeat the washing four or six times for thorough cleansing of the viscus.

In concluding the question of lavage, Professor Fleiner's (44) dictum may be quoted: "If nothing injurious is introduced into the stomach, less will be brought up by lavage; by a proper diet, therefore, we may limit lavage and in many cases make it unnecessary."

*Massage and electricity.*—General massage, by improving muscular and general metabolism, is decidedly helpful, especially in those cases where confinement to bed is necessitated. Massage localised to the region of the stomach, however, should only be prescribed with caution, as unless the operator is specially skilled in abdominal manipulations the dilatation may easily be aggravated instead of benefited.

Mechanical vibration or sismotheraphy is generally of greater therapeutic value than ordinary massage, since it is capable of more delicate and varied as well as more vigorous and sustained application. In suitable cases it certainly improves both the secretory and motor functions of the stomach.

The vibration should be performed over the region of the viscus with a round flat applicator about two and a half inches in circumference, beginning with five minutes and gradually increasing to ten to fifteen minutes once a day. I have obtained excellent results from this method in cases of moderate severity, it being particularly helpful where epigastric tenderness and pulsation is a marked feature.

The value of electricity in atonic dilatation is now fully established, chiefly owing to the work of Boardman Reed in America and Herschell in England. The various forms of electricity which have proved beneficial are the galvanic, faradic, galvano-faradic, mono-phase sinusoidal, triphase sinusoidal, and high-frequency currents.

It may be said at once that high-frequency

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currents are of value simply from their general tonic action, as they have no power of causing muscular contraction. They are, therefore, chiefly of value in mild cases.

The other forms are useful in all stages of atonic dilatation if properly applied.

It must be borne in mind, however, that whatever form of electricity is used marked benefit cannot be looked for in severe cases without the aid of an intra-gastric electrode, so that the current may come into direct contact with the walls of the stomach. The intra-gastric electrodes in general use are Einhorn's, Boardman Reed's and Boas's. I have used all these but prefer that of Boas. This consists of a wire spiral enclosed in a narrow rubber tube with several small lateral openings at its distal end and a fitting for a rheophore at the proximal end. The tube is passed in the same manner as a lavage tube, the patient having previously sipped half a pint of water for the electricity to make contact with the walls of the stomach. If preferred, however, the water may be introduced through the tube.

With regard to the relative value of the various forms of electricity mentioned, the triphase sinusoidal current is certainly pre-eminent. It was first introduced as a therapeutic agent ten years ago by Guimbail of Paris, Herschell being, apparently, the first to use it in England. He claims that (45) "the triphase alternating current appears to be *par excellence* the current to use for the purpose of restoring tone to the gastro-intestinal tract, producing manifest contractions in the stomach and intestines and considerably shortening the time that food remains in the stomach." After nearly two years' experience of this form of electricity I can fully endorse the above statement.

*Hydrotherapy.* — Of the many hydropathic methods advocated for dilatation "the Scotch douche" and "the Priestnitz pack" are probably the most beneficial.

In "the Scotch douche," or rather, the modification of it, the patient lies in a bath containing enough hot water to cover the whole of the body with the exception of the abdomen. Jugs of hot and cold water are then poured alternately upon the epigastrium for three or four minutes from a height of three feet, gradually increasing this distance to five or six feet. This acts as a powerful stimulant to the vaso-motor system.

"The Priestnitz pack" consists simply of a folded towel dipped into hot or cold water, wrung out and applied to the epigastrium. It is then covered with oiled silk and the whole held in place by a broad flannel bandage passing round the body. It certainly greatly relieves pain, but how the effect is produced is a matter of conjecture.

### Surgical Treatment.

Whether surgical interference is ever justifiable in atonic dilatation is a moot point, for it is doubtful whether the results obtained are good enough to justify the risk of adhesions and other complications which may follow a laparotomy.

Gastro-plication and gastro-enterostomy are the chief operations which have been performed. Gastro-plication seems to have but little to recommend it, since although it reduces the size of the viscus it does not cure its atony. Gastro-enterostomy, on the other hand, by preventing stasis may possibly enable the viscus ultimately to regain some of its muscular tonus.

### General Hygiene.

This includes such self-evident advice as to have the teeth put in good order if defective, to eat slowly, masticate thoroughly and to rest before and after meals, preferably with the foot of the bed or couch raised, so that the stomach may be saved the strain of gravitation.

In severe cases it is helpful to wear an abdominal belt or supporting bandages, whether gastroptosis is present or not.

As to climate, mountain or high moorland air is preferable to that of the sea.

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*September 21st, 1908.*

# SCOPOLAMINE-MORPHIA AND CHLOROFORM ANÆSTHESIA.

By H. MACNAUGHTON-JONES, M.D., F.R.C.S.

I HAVE used the scopolamine-morphia-chloroform method of anæsthesia in a number of serious cœliotomies since my return from Professor von Krönig's clinique in Freiburg at the close of 1906, and find that it has been resorted to in twenty-eight cases at St. Ronan's Medical Home. I have specially reserved it there for those operations in which I anticipated that prolonged anæsthesia would be demanded, and in several instances where the condition of the patient was such as to make me anxious regarding the low vitality and the power of resistance to the shock of operation.

These operations included 3 hysterectomies, 2 appendicectomies with pelvic complications, 12 salpingo-oöphorectomies or resections of the ovaries, some prolonged vaginal operations, and two very extensive post-operative herniæ. The ages of these patients were: Over 70, 1; 60–70, 2; 50–60, 4; 40–50, 7; 30–40, 10; 20–30, 3; under 20, 1. The duration of the operation in 3 cases was over two hours, in 6 over one and a half hours, in 5 over one hour, in the remainder under the hour.

I have also made use of this method in a few operations other than gynæcological, as in the case of a young lad whose pulse was habitually 48 to the minute and who was very anæmic. Here abdominal exploration revealed diseased glands, and adhesions encircling the intestine. The pulse rose to 55 during the operation, but there was not the least shock from it, nor any sickness afterwards. Again, in a lady for amputation of the breast, who had been operated on some years before for carcinoma, and had general disséminated carcinoma of the glands and skin nodules. One of her lungs was practically useless. She had a compressible and rapid pulse. The operation lasted forty minutes. There was not the least shock, nor any sickness. I have not referred to details of the other operations, but perhaps I may mention a few to show the character of some of the cases in which I used scopolamine.

A patient, æt. 65 years, was brought after a long journey to London in such an exhausted condition from pain and discharge that an examination at the

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time was impossible. She was sustained with strychnine injections and by rectal feeding for some days before I ventured to operate. The growth was a columnar-celled carcinoma of the vagina. The operation lasted an hour. She made an excellent convalescence, leaving the home fourteen days after the operation.

A patient, æt. 39 years, weighing barely five stone, had a kidney displaced into the inguinal region, and a dermoid cyst of the left ovary. The kidney was anchored by the lumbar incision, and the dermoid cyst removed by abdominal cœliotomy. The time occupied in the two operations was fifty-two minutes. There were no post-operative effects.

Three cases operated on under this method died in the Home. The first, æt. 23 years, was brought in with a tympanitic abdomen and obstructed bowel. The condition at operation was proved to be due to a solid and inoperable growth, which completely blocked the lumen of the rectum and involved the sigmoid. The second death occurred from jaundice and phlebitis in a patient with enormous varicose veins in both legs, and from whom a large semi-solid ovarian cystoma was removed. She had previously suffered from repeated attacks of phlebitis and gall-stones. The symptoms did not supervene until after the stitches were removed on the sixth day. The immediate recovery from the operation was excellent. The third death occurred in a patient admitted in a practically hopeless condition, suffering from (as proved by exploration) inoperable carcinoma of the omentum and adnexa. There were no symptoms due to the exploration.

For some years, since reading the experiments of Professor Schäfer and Mr. Scharlieb, I have given an injection of strychnine—one sixtieth to one thirty-sixth of a grain, and atropine—one hundredth of a grain, an hour before operation in all cases of chloroform anæsthesia for any serious operation. The physiological effect on the pulse fully bears out the conclusions they arrived at as to the influence on the blood-pressure. In the great majority of the cases I have had administered the night before operation one sixth to one eighth of a grain of morphia and one hundredth of scopolamine. The following morning (operating at 9.30 a.m.), when the bowel has been finally and thoroughly emptied, a second injection of the same quantity is administered, and from half to one hour before operation the strychnine and atropine is given. In every instance save one the chloroform was administered with a Vernon Harcourt's regulator.

There is no necessity to refer to the influence of the scopolamine and morphia addition to ordinary chloroform anæsthesia. This has been so repeatedly written on that it is universally understood. In some of the lighter cases a morning injection only has been given, and in some strychnine alone without the atropine beforehand.

I have never resorted to spinal analgesia, as I am quite satisfied with the general method. When in Heidelberg and Freiburg in 1906, I was surprised to find that every gynæcological operation was performed under spinal anæsthesia, and with complete success — in the latter clinique with the additional use of scopolamine and morphia.

In my own operations, the patient is brought into the theatre in a quiet and tranquil state, sometimes slightly narcotised; she quickly goes under the chloroform and remains in a most satisfactory condition during operation. The post-operative results as regards immediate pain, sickness, and general discomfort are, as a rule, better. With regard to sickness there are exceptions, and in these latter the vomiting is not influenced. There are no disagreeable post-operative effects. Only in one case, that of an abnormally obese woman with weak heart action, in which the abdominal incision went through nearly three inches of fat, had the chloroform to be discontinued for a time. This operation, which was a most severe one owing to serious bowel complications, was completed in two hours, and there were practically no after consequences.

I have asked Dr. Dudley Buxton, Mr. Scharlieb, and Mr. Bakewell, who have mainly anæsthetised the patients at St. Ronan's, to write me a few lines giving their experience generally of the results: Dr. Dudley Buxton says: "Through the courtesy of Dr. Macnaughton-Jones I have been able to adopt this method in a number of cases, and have also employed it on various other occasions. The character of the narcosis differs from the familiar combination of chloroform and morphine. The patient passes very quietly into what resembles placid sleep. Even when the conjunctival reflexes are not abolished, true anæsthesia appears to be present, and muscular relaxation complete. There

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is no stertor, but if the chloroform is pushed, duskiness supervenes with slow and shallow respiration. As I have worked in all cases with Vernon Harcourt's regulating chloroform inhaler, I have been able to mask this condition, and lessen the percentage of chloroform. The amount, whether judged in percentage or in bulk, required for even severe operations, is very small. If more than the minimal percentage or amount is given, the profound and, I think, dangerous degree of narcosis supervenes. I habitually use oxygen with chloroform, and it is very valuable in the sequence I am discussing. In nervous excitable women the antecedent injection of scopolamine and morphia is most useful. It has the effect of inducing a calm, happy and hopeful state, and they inhale the general anæsthesia more easily and readily. The dose of morphine is important, and should be varied accordingly to the general state and physique of the patient. As to after-effects, they are, I think, less severe. The dangers are those familiar to surgeons who have employed morphine and chloroform. To avoid them the utmost care is necessary to limit the amount of chloroform used, and as very little of this is really needed this is readily done, especially with a regulating inhaler. The patient should not be pushed to the depths of chloroform narcosis essential when that anæsthetic is used alone, as the danger arises from paralysis of the respiratory centre. Fortunately, the gradually deepening narcosis and lessening of depth of respiration, with the duskiness of colour that appears, give timely warning of any peril." Mr. Herbert Scharlieb says: "It is impossible to argue from a few cases, but judging from those I have seen I should say that patients under the influence of scopolamine and morphia need less chloroform to anæsthetise them than those who have not had a prior injection of these drugs. I have never seen any ill-effects in patients to whom scopolamine and morphia have been given, and I do not think that the modification of the usual conditions of the pupil during anæsthesia matters in the least." Mr. Bakewell says: "I have only given chloroform by the scopolamine and morphia method for Dr. Macnaughton-Jones. Most of the cases have been cœliotomies, and in some instances as severe operative procedures as I can well imagine. There has been a curtailment of the initial stages of the anæsthesia, and the total quantity of the chloroform administered has been smaller. There was less excitement during the induction period. I have never seen any alarming symptoms arise during administration by this method."

*September 21st*, 1908.

# A CASE OF

# CARCINOMA OF BOTH BREASTS.

By W. MORLEY WILLIS, F.R.C.S.Eng.,
Surgeon to the Children's Hospital, Nottingham; Senior Assistant Surgeon, General Hospital, Nottingham.

In spite of the enormous amount of work which has been and is being done in the field of cancer research I think it true to say that we know nothing of the essential causes of the disease. Beyond the facts that the disease is a development of one or the other of the types of epithelial cells which occur in different situations of the body, and that such cells develop and grow in places where they are not normally found, that they tend to be disseminated along the lymph channels to places more or less remote from the original source, we don't know what cancer really is.

In time, for some reason or other, the development and growth of these cells in their abnormal situations produces a gradually increasing prejudicial effect upon the health of the individual, and ultimately kills.

In relation to treatment of the disease, as a result of research work of numerous people matters have, however, vastly improved.

Taking breast cancer as an illustration; it was long ago discovered that cancer cells had permeated the lymph channels and were present in the glands of the axilla before there was any appreciable enlargement of the glands to be detected; and thus for many years it has been the rule to make a clean dissection of the axilla in all cases of operation for cancer of the breast.

This, later, was shown to be inadequate; and now I suppose it may be said that the rule in removing a breast for cancer is to endeavour to remove the entire lymph channels and glands which issue from the breast, and are capable of being infected.

This involves removal of both pectorals, carrying the dissection from the clavicle down to the level of the ensiform cartilage so as to remove the aponeurosis covering the upper part of the rectus abdominis, and also going beyond the middle line to the opposite side of the sternum, and to the opposite rectus abdominis in its upper part.

All this, with a considerable area of skin, is

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removed in one large mass, including the axillary contents, so as to prevent as far as possible the liberation of cancer cells into the wound thus made by any incision into the growth, or by any division of a lymph channel permeated with cancer cells.

The result of all this is that at the present day operation for cancer of the breast shows a very considerable proportion of cures, and longer delay in recurrence where cure is not effected, much saving of life being attained.

As our only means of treating cancer is by operation, it is a matter of the greatest importance to recognise the disease at the earliest possible moment, and I think that in this direction an improvement has taken place.

The public have become aware of the improved results of operation, and the importance of early operation, and patients more promptly consult their doctors than formerly, and so the cases reach the operating surgeon earlier in the course of the disease.

It seems clear to me that it will never be possible to diagnose cancer at its very beginning, consisting as it does of one cell (and it may be only one) getting "out of bounds" so to speak.

I don't suppose anyone ever proposed to remove the vermiform appendix in a perfectly healthy individual who had never had an attack of appendicitis—though this may have been done.

It is certain, however, that no one would propose to remove a breast because of the possible development of cancer if the breast were apparently perfectly healthy.

There are conditions which arise in various organs sometimes, and which have been regarded as favouring the development of malignant disease.

These have been termed "pre-cancerous," and amongst them is numbered chronic interstitial mastitis.

The case which forms the subject of this communication there is reason to believe commenced in this way, and it possesses features which make it worth considering.

Mrs. H—, æt. 39 years, consulted me first on December 19th, 1904, at the General Hospital complaining of some trouble with the left breast. My note on that day is as follows:

"Tender, diffuse swelling of the left breast, nipple retracted. No definite lumps to be felt, but an induration of the whole breast.

"On lifting it up it is tender, but not when pressed upon with the flat hand. No glands in the axilla to be felt."

The condition had been noticed by the patient for about five months, and at the time she first saw me she believed herself pregnant (two months). In my book I made the note "? Chronic mastitis," and she had some treatment for this condition.

On January 2nd, 1905, I made a note that there was no swelling but an induration of the whole gland—chiefly above the nipple.

The breast was still tender when pinched up, but not when pressed on with the flat hand.

January 30th, "Still no suggestion of a tumour; breast smaller and less tender."

She was seen again by me in March and April, 1905, and the state of affairs remained much the same. I advised, however, that she have the breast removed, but she preferred to wait until her pregnancy was over and her baby born (this event occurred in July, 1905).

My friend and colleague, Mr. A. R. Anderson, saw her with me on one occasion, and he shared my view as to the diagnosis (chronic interstitial mastitis), and also advised the removal of the breast.

In February, 1906, she was admitted into the General Hospital, without my knowledge, and came under the care of a colleague who was unaware of my interest in the case.

He independently formed the opinion that the condition was one of chronic mastitis, but he did not advise any operation, and so she was discharged next day.

I now lost sight of her for nearly two years, until early this year she came to the hospital again to see me.

The left breast, the one originally affected, showed very little change. There was no definite tumour to be felt, but an induration of the whole gland. There were now distinctly palpable enlarged glands in the left axilla.

The greatest point of interest lay in the fact that the right breast was now the seat of disease also, and this (she said) had been coming on for about twelve months.

The condition was similar in every way to that which was first noticed in the left breast, viz. no definite tumour, but a diffuse induration of the gland, which was slightly tender. The skin in

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neither case was adherent, nor was either gland adherent to the underlying fascia. The nipples were each retracted. Enlarged glands were to be felt also in the right axilla, though not so marked as in the left.

She was admitted into the hospital in May, 1908, and I removed both breasts and the contents of each axilla, a fortnight's interval elapsing between the operations.

I did not, as is my usual custom in operating for carcinoma, remove the pectoral muscles.

The fascia overlying the pectorals was cleanly dissected off, and the breast and axillary contents completely removed in one mass on each side. She was discharged, with both wounds soundly healed, in exactly a month from the day of her admission, and went to the convalescent home.

Each breast was examined microscopically by my friend Dr. F. H. Jacob, and in each case the primary growth and the glands showed carcinoma.

This case opens up various interesting points which might be discussed.

First, was the left breast really chronic interstitial mastitis, or was it carcinoma from the very beginning?

This is, of course, quite impossible to answer dogmatically, but the absence of a definite tumour, and the diffuse character of the induration in this breast, the tenderness, the non-implication of the skin or underlying structures, all made it probable that it was chronic inflammation.

Then another point is the question of the development of disease in the right breast. Was this carcinoma from the beginning, and was it a direct dissemination of this disease from the left breast?

My own view is that the left breast was originally, and when I first saw it, the seat of chronic interstitial mastitis; that in this breast carcinoma developed; and that the right breast was carcinomatous from the commencement of disease in it, and that this was brought about by direct continuity from the left breast.

Had I been quite sure that each breast was carcinomatous at the time of my operation I should have removed the pectorals in the usual way, leaving only the clavicular portion of the pectoralis major.

This case more than ever confirms me in my opinion that chronic inflammatory affections of the breast, which do not after a period of four or six weeks show signs of improvement, should be removed; and that it is safest to remove swellings of the breast the nature of which is doubtful.

*September 21st*, 1908.

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**Chronic Pancreatitis.**—J. B. Deaver says the most frequent form is the interlobular, and that it is this with which we have to deal in the pancreatitis complicating disease of the biliary passages. The interacinar form is due to systemic conditions not yet understood, and is correspondingly less amenable to direct or local attack. The most frequent cause is doubtless some obstruction to the free discharge of the pancreatic secretion, accompanied or not with an ascending infection. Either factor alone is enough to produce it. Many other causes of chronic pancreatitis have been claimed, cardio-renal disease, tuberculosis, syphilis, etc., and Deaver thinks that microscopic examination would reveal many cases of beginning change in normal appearing organs. The local symptoms are not very significant and may be practically absent; the examination of the fæces is sometimes inconclusive, and demands skill not always easy to command. The symptoms depending on the derangements of metabolism, due to interference with the internal secretion of the pancreas, are most distinctive. Diabetes is often too late a symptom to be of practical value, but the Cammidge reaction is a fairly constant sign of pancreatic disease. Medical treatment is generally futile, but by surgery we can in many cases remove the underlying cause, overcome the infection, and bring about complete recovery. When secondary to gall-stone disease, removal of the calculi and free drainage will, in most cases, lead to a complete subsidence of pancreatic symptoms. When the gall-ducts are found clear, and the causal infection has subsided, drainage of the organ by way of the biliary passages nevertheless exerts a remarkable curative effect on the lesion, especially when the diagnosis and operation are made early, before the so-called catarrhal pancreatitis has really become a chronic interstitial interacinar lesion. Moderate anæmia and glycosuria are not contra-indications, as both are often greatly benefited when normal metabolism is restored.—*Medical Record*, vol. lxxiv, No. 7.

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# ON EXPLORATION OF THE INGUINAL CANAL.

A Clinical Lecture delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—More than ten years ago I began to advise that the inguinal canal should be explored. As you are aware, I do not look with a very friendly eye upon exploratory operations. The fact of a surgeon undertaking to perform an exploratory operation implies that he has not made a diagnosis. But several things have happened which have led me to regret that I have not explored the inguinal canal oftener. Last year a little boy was admitted into Lucas Ward who had a rupture upon the left side. Although he was young, perhaps younger than we generally operate, yet there were reasons why it should be undertaken. Perhaps one of the reasons was this: that he came from South Africa, where trusses, perhaps, are not very easy to obtain, and where, perhaps, trusses are not very easily worn, and where skilled assistance is not always available should the rupture come down and become strangulated. So that his left side was operated upon and he speedily got well. He had not been very long out of the hospital before he had some occasion for straining and a rupture came down from the right side. He then was admitted into the hospital again, and the rupture on the right side was cured. I need hardly say that when he was examined before his first operation our first attention was directed to the right side, but we could discover nothing there which would lead us to suppose that he might develop a rupture. But nevertheless he did so. I regret that the right inguinal canal was not explored at the time of the first operation. The opening of the right inguinal canal would have been the most trivial addition to the pains or perils of the

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operation. Indeed, I believe this: that unless the patient was told that an operation had been performed on the other side he would hardly be conscious at all of the fact. You might say that an exploration of the right inguinal canal, which, of course, necessitates the division of the aponeurosis of the external oblique, would have weakened that canal and made him liable to a rupture, even if none had been found. But this I do not believe. My experience teaches me that there is hardly any structure in the body which unites more firmly and strongly than the aponeurosis of the external oblique if the parts are properly brought together and given a proper time for repair. Another occurrence has led me to regret not having explored the other inguinal canal whilst operating upon one side of the body. Three years ago a gentleman returned from India, and on that occasion he had a rupture on the left side. Radical cure was performed, and there was no peculiarity in his case except that at the time of the operation I observed that he had an unusually long slit in the aponeurosis of the external oblique, leading up from the apex of the external abdominal ring. After his operation he went back to India, where he pursued all sorts of sports and athletic exercises and, as he said, tried my operation very hard indeed. However, that side remained right. But towards the end of last year I was informed that the operation had been unsuccessful, and that he was returning from India to have it done over again. I never attach importance to anything I am told about a patient who has had an operation performed, but wait until I have seen him. Possibly he has some new trouble. In a day or two this patient arrived, and then it appeared that he had developed a rupture on the right side. He had to have this operated upon because he wished to return to India. People who live in India cannot easily wear trusses. At some periods of the year the climate is so hot that the wearing of a truss becomes almost insupportable. In addition to that trusses are not a certainty; they occasionally fail. Then if they do fail and the rupture comes down and is strangulated, in a country like India skilled assistance is not always available. These were perhaps some of the reasons which led this gentleman to have his operation performed again on the right ride. He had one further reason, which was that he had had experience of the great benefits of radical cure upon the left. At the time of his second operation the canal was opened and then we found a sac which was obviously congenital, and probably the sac of an ordinary congenital funicular hernia. Those two cases certainly made an impression on my mind, and I do not know why I did not on the first occasion explore both canals.

Now I will try to proceed to convey to your minds some of the reasons which would lead me to explore the inguinal canal, and first of all will refer to the pain at the situation of the internal abdominal ring. Of course I cannot tell you how they describe this pain which indicates the commencement or the presence of a hernial protrusion. First of all they say that when they wake up in the morning they are comfortable, but that after walking about for a part of the day the pain comes on and the seat of the pain is indicated as the internal abdominal ring. And on proper examination you may see that they have at that place an impulse. Observe I said you may *see* that they have an impulse. The impulses of which I am speaking are very slight. I do not believe that they are impulses which you can feel, but if you stand the patient up in a perfect light and then proceed to use your eyes, you may observe a very slight elevation at the situation of the internal abdominal ring, and when they cough you will see a small localised impulse. I attach great importance to that. Of course many people's abdomens heave and show a general impulse when they cough, but the impulse of which I am speaking is a localised impulse, which is most difficult to detect. I have over and over again explored an inguinal canal simply because the patient has complained of pain and because I have seen this minute and almost imperceptible impulse. And when I have done that I have made some curious discoveries. I remember a lady who had seen a great many surgeons because she had pain in the left inguinal region. She had had a great many opinions given concerning her case, and in consequence she had got into a condition of great perplexity. I think there was no question about the reality of her pain. I felt sure that she had a pain, and she gave a typical history, namely, that she had not got it in the morning, but that it came on towards the close of the day. When she stood up and was scrutinised in a bright light, a little eleva-

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tion was seen over the situation of the internal abdominal ring. Furthermore a little impulse was seen there, and therefore I advised her to have her inguinal canal explored. We then found in the upper part of the inguinal canal a considerable piece of fat, and in the centre of that fat a hernial sac which I may describe as sufficiently large to admit the first joint of an index finger, and it was treated in the usual way. Again, I recall the case—and I am merely picking out typical cases—of a little boy, the son of a gardener, who was admitted into the hospital because he had a hernia on the left side. That hernia possessed the usual characters, and he had a large hernial sac with a wide mouth. But on the opposite side he had the faintest possible impulse in the inguinal canal. We therefore opened that inguinal canal because of this minute faint impulse, and we found at the upper part of it a small hernial sac. I cannot help thinking that had we not opened the inguinal canal the probabilities are that this boy would have ultimately developed another rupture.

I am now coming to another reason which has on a great many occasions led me to explore the inguinal canal. I have not had time to go over the very large number of cases of which I have a note, and to tell you how often I have been deceived by the indication which I am about to mention. But my recollection is that the cases in which nothing has been found are very, very rare. The following is the ordinary history of the case in which the canal is explored, because of a patulous condition of the external abdominal ring and of the canal itself. A young gentleman was placed under my care because he wished to enter one of the public services. On examination it was found that on the right side the external abdominal ring was capacious and that the finger could be passed into the canal. I will pause here for one minute to tell you that you must have in your minds some concept of the proper and normal condition of an inguinal canal. When the finger is passed up the neck of the scrotum in a proper manner, the tip of the finger feels first of all for the spine of the pubes, and above that it feels for the external abdominal ring. I think a perfect external abdominal ring will feel tight all round. You can feel its edges all round, and I do not believe you ought to get more than just the pulp of the tip of your index finger into that external abdominal ring. Next, when the patient coughs and strains, that small taut ring ought to contract and extrude the tip of the finger. If you can go further and get the tip of the finger through the external ring into the canal, there will probably be a hernial sac there. And if you can pass your finger into the ring and into the canal, and then push it backwards and feel you are entering the abdomen, you can be sure that that is a very defective canal, and that that patient is almost certain to have, or is certain ultimately to develop, a rupture. In the case of this young gentleman, the finger entered his external ring and entered his canal, so that it was explored. And within it was found a small empty hernial sac. It is clear that if, when operating on the right side of this young man and curing his right-sided hernia, I had omitted to explore the left side, which had a patulous ring, I should have left him with a hernial sac and he would almost certainly have developed a rupture. I might proceed to tell you of a very large number of patients in whom I have explored the canal simply because the external ring was enlarged, and if I had the opportunity of tabulating them all it would be found that it was the rarest possible thing not to find a hernial sac in the inguinal canal.

I am now coming to another class of case in which it is advisable to explore the inguinal canal. And that is in cases of scrotal and inguinal varicocele. When you examine a patient who has a scrotal varicocele, you may remember that I advised you not to be content with assuming you have to deal merely with a scrotal varicocele, but to examine very carefully the neck of the scrotum. In cases of varicocele you not infrequently feel a thickening of the veins passing up into the external abdominal ring. And next, I have advised you strongly never to omit to examine the inguinal canal yourself. The enlargement of the veins in a great many instances passes up through the external abdominal ring, which is almost invariably dilated, and passes inwards into the canal, which dilates too. I believe it is this expansion of the inguinal canal by these enlarged varicose veins which causes this condition to be so often complicated by the presence of a hernial sac. I have only to qualify what I am saying in one small particular, and it is this: that the sac is very often a small sac, which has very often a very sharp

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point to it, which causes one to think that it is not necessarily formed by a protrusion from within.

Here is a typical instance illustrating the importance of exploring the inguinal canal in cases of scrotal and inguinal varicocele. A gentleman who was occupied in one of the public services was afraid that he might be rejected because on the left side he had a very large scrotal varicocele, and I ask you particularly to observe that although he was under the care of one of the most competent practitioners that I know, this was the trouble for which he was sent. He was sent because he had a scrotal varicocele. However, when I came to examine his varicocele I found that he had dilated veins in the neck of the scrotum. Next, I found that the external abdominal ring was patulous, and I could easily get my finger into it. Next I observed that he had a very slight impulse in his left inguinal canal. The course to pursue was obvious. The operation was performed through the inguinal canal. Dilated veins were met with, but in addition to that he had a small hernial sac. The medical man who was associated with me in the case was perfectly candid. He said that he had not in the least appreciated the condition. I think if he himself had undertaken that operation he would have performed the ordinary scrotal operation for varicocele; he would have overlooked the dilated veins in the inguinal canal, and he certainly would have overlooked a hernial sac in that canal. And I think it is highly probable that that patient would afterwards have developed a rupture. I will make a remark in passing about the way in which that operation was performed. Of course the inguinal canal was opened, varicose veins were separated from the vas deferens and from the spermatic artery, and then they were clamped above and below, the intervening piece was cut out, and now observe what I shall tell you. We treated the pedicles which were left as you must treat every pedicle. We transfixed and tied them; and I warn you that if you do not transfix and tie these pedicles in operations for varicocele, or, for the matter of that, for the removal of hernial sacs, there will be occasions upon which ligatures will slip and you will be confronted with a most troublesome and dangerous condition of things. It is a golden rule in surgery to tranfix all pedicles. Whilst I am speaking on the subject of varicocele I would draw your attention again to the expression "inguinal varicocele." Of course you are aware that a patient may have a varicose condition of the veins of the inguinal canal without any varicosity of veins in the neck of the scrotum or in the scrotum itself. And that is a very puzzling condition, and it is one which is often overlooked. An inguinal varicocele may cause considerable pain by distending the canal, and be associated with an obvious impulse, so it is easy to understand how they are often mistaken for a rupture. It is not a very serious mistake to make, because it may merely lead to the patient being supplied with a truss. However, I have observed that patients are unable to wear trusses on inguinal varicoceles; the dilated veins are almost invariably too painful to allow of the pressure of a truss, so that patients may needlessly wear trusses and may needlessly have a good deal of pain and discomfort inflicted upon them.

I have the notes of one case in which both inguinal canals were explored in a youth who wished to join the South African police. My note says that the right side was empty, and that the left contained an inguinal varix. So I am able to give you one of the few instances in which I have opened the inguinal canal needlessly. However, that inguinal canal is perfectly repaired, and I have no doubt that a few months after, except for the scar of the operation, no one would be able to tell it had been done. I might give you instances of inguinal varicocele in which the varicocele had been associated with a large amount of fat, constituting what is called a lipoma of the cord. I have mentioned another indication for opening and exploring the inguinal canal, and I need hardly refer to that again. And the indication is that when a youth has a rupture of one side of the body, you must consider very, very carefully the desirability of opening the opposite inguinal canal. My own experience teaches me that in many instances it is better to explore that inguinal canal than to leave it. I do not know, but I suspect that you will very seldom fail to find a sac in the opposite canal.

I am now coming to another class of case in which it will be advisable for you to explore the inguinal canal, and that is in cases of congenital hydrocele. This is the ordinary history of a case of congenital hydrocele. And I would say this, that you will find in practice that congenital

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hydroceles are a great source of trouble, both to the medical man and to the parents. The infant is observed to have a swelling in the scrotum; the swelling is translucent. It may be flaccid in the morning, but it becomes tense during the course of the day. But perhaps diligent pressure will cause some return into the abdomen. But I do not know that you can attach very much importance to that. The cord in the neck of the scrotum may be feeling a little thickened. I doubt whether you will see impulse in the inguinal canal. Impulses in babies are difficult to detect. The ordinary routine treatment of such a case is to first of all supply the infant with a small rubber-covered truss. I advise you to give a double inguinal truss, one which makes pressure on both sides. My reason for that is simple. A double inguinal truss is very easy to adjust, and is very well borne. And the opposite side, as you will have observed, is always an object of suspicion. The truss is given because there is a patent condition of the funicular portion of the processus vaginalis, and you hope that the pressure of the truss may lead to its closure. The next treatment is usually to order a little chloride of ammonium lotion to be applied to the scrotum. The ordinary lotion is fifteen grains of chloride of ammonium, a drachm of rectified spirit, and water to one ounce. I do not know how it acts, perhaps as a slight irritant, and may cause some contraction of the scrotum. It is occasionally efficacious. Supposing that after a time the application of chloride of ammonium fails, the next step will be to tap the hydrocele with a capillary trocar, the Southey's trocar, an excellent surgical instrument invented by a physician. That will probably lead to a cure of the hydrocele, but the child should wear the truss until it is two or three years old, and even then be kept under observation to see that nothing further develops. But if the hydrocele does not get better an operation should be done. And the operation which I perform is to open the inguinal canal, and look to see what has happened to the funicular process of the peritoneum, which is very delicate and not easy to find, and then to pull up the tunica vaginalis through the wound and remove a portion of it, so that no fluid can collect in it. If the patient had a hydrocele you will seldom fail to discover that the funicular process of the peritoneum is still patent.

I now come to another class of case in which it is desirable to perform exploration of the inguinal canal, and that is in cases of ill-descended testes. Here is an ordinary case. A boy has his testis situated just outside the external abdominal ring. Leading to it can be felt the spermatic cord, hard, but apparently with nothing in it. In the particular instance to which I am referring there was not any undue patency of the external abdominal ring or of the inguinal canal; but the mere fact of that boy having a developmental defect in his testicle should lead you to suspect that he may have other developmental defects, and that his funicular portion of the processus vaginalis may be patent. In the particular instance of which I am speaking the canal was explored and a funicular sac was found. Another case of ill-descended or improperly descended testis in which the canal has to be explored is that in which the testis in its transition has passed down the canal, out at the external ring, and then passed backwards over the external oblique so as to lie over the inguinal canal. That is a very awkward place for a patient to have a testicle. It is usually said to be painful, and is not infrequently associated with either rupture or, as in the case which is before me, with hydrocele. Supposing you were to operate upon a testis which lay in front of the canal with the object of passing that testis into the scrotum, you might find a well-developed testis and tunica vaginalis, and with a cord which might apparently be right, and with an external ring and a canal which might not appear to be obviously wrong. However, in cases of that sort I have made it a rule to open the inguinal canal to see what was inside it I am speaking from memory, but invariably you will find they have inside a patent processus vaginalis, and that had the canal not been opened they would have been liable to the descent of a hernia.

There is one other consideration which has led me to explore the inguinal canal. Not infrequently patients are sent who are said by their medical man to have had a rupture, and want to have a radical cure of hernia performed. I must say that I do not myself like performing operations on diagnoses which have been made by other people, and our routine in cases of this sort is to make the patient walk about without a truss and see whether the rupture comes down. In quite recent times

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we have had a man under our observation who was said to have had a rupture on the left side, and who had worn a truss. We could discover no sign of hernia. The external ring was small, there was no impulse in the canal, and I should have said, had I not known the medical man, that he had no rupture at all. However, the medical man was a friend of mine, and I knew he was capable, and therefore I decided to explore that inguinal canal. When we did so, we found in it a hernial sac which was at least one and a half inches long, and capacious.

I daresay there are other reasons why the inguinal canal should be explored, but I have given you, I think, most of those which occur to my mind. And I have more often had occasion to regret not having explored the inguinal canal than to regret having done it.

*September 28th*, 1908.

**Embolism of the Lungs after Radical Operation for Inguinal Hernia.**—Mauclaire reports a case of bilateral phlebitis of the spermatic vein and slight pulmonary embolism after an operation for inguinal hernia on a man aged 42. He summarises twenty-five cases in the literature, with twenty-five others briefly mentioned, and seven cases of femoral phlebitis. The mortality in the twenty-five cases of which the details are known was nearly 50 per cent. The embolism is usually tardy, of sudden onset, and is generally accompanied by hæmoptysis. Prophylaxis consists in avoidance of injury to the vessels in the epigastrium and of traction on the femoral vein and manipulation of the vessels in the spermatic cord. Injections of serum and copious drinking of water have been recommended to prevent this post-operative embolism, on the assumption that the patient is dehydrated. If phlebitis develops in the spermatic vessels the region must be immobilised, and he suggests that embolism might be prevented by applying a ligature high on the spermatic vein. He has had considerable experience with this intra-abdominal ligation of the spermatic vessels, and he no longer hesitates in very urgent cases to ligate the femoral or iliac vein in prevention of embolism. In regard to treatment of femoral thrombosis and pulmonary embolism, he merely calls attention to Trendelenberg's recent attempts to remove the obstructing clot after resection of the third rib and arteriotomy of the pulmonary artery.—*Journ. A. M. A.*, vol. li, No. 7.

## THE RÔLE OF GASTRO-ENTEROSTOMY IN GASTRIC ULCER AND GASTRECTASIA.

Post-Graduate Lecture delivered at Ancoats Hospital.

By E. STANMORE BISHOP, F.R.C.S.Eng.,
Gynæcological Surgeon, Jewish Memorial Hospital;
Hon. Surgeon Ancoats Hospital, Manchester.

GASTRIC ulcer has attracted much attention in the surgical world of late owing to the fact that the operation of gastro-enterostomy appears to exert a very favourable influence upon its course. This has led, on the one hand, to increased scrutiny of the symptoms already known which indicate its presence and search for others, and, on the other hand, to improvements in the technique of the operation itself, so that it may produce the best effect, and be free from certain drawbacks which followed the earlier methods.

Diagnosis in ulceration of the stomach is surrounded with difficulties even in advanced cases, whilst the earlier stages have many things in common with other less serious complaints.

Constant and repeated examination of histories of cases proved by operation or autopsy to be true examples of gastric ulcer would seem to be the method which promises to be the most fruitful of results. Such comparison has so far resulted in this, which may be taken as very nearly a typical history.

For some years, or possibly months, before a patient applies for relief he or she has suffered from a sense of weight or pressure about the epigastrium, which has been noted as being at its worst soon after a meal, especially after certain articles of diet which he has learnt to avoid. These things are usually those which excite free secretion of gastric juice, such as butcher's meat, or which contain undigestible vegetable fibre, as cabbage, and which therefore remain for a longer time than normal in the stomach. This sense of weight is relieved when the food has passed out of the stomach into the intestine. But the discomfort, which at first was only occasional, has become more constant, so that he almost dreads to take food because of the inevitable result; moreover, he has found that pressure of tight clothing around the epigastrium is not pleasant, and he has taken to wearing looser waistcoats, or, if the patient is a woman, she has

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had to discard the wearing of corsets, at least after a meal.

Vomiting may have occurred, or there has been eructation of very sour material into the throat; both about the time when digestion is at its highest, that is, about one and a half to two hours after a meal. If vomiting has been actual, relief has been at once obtained, though the teeth have been set on edge by the acid character of the vomit. In some cases the vomiting occurs first thing in the morning when no food but a cloudy very acid fluid is brought up; the vomit contains no visible blood.

Or he may have found that he can obtain relief from the gastric pain by eating white of egg boiled, which unites with the free acid in the stomach, or by taking alkalies, occasional does of chlorodyne, or Mother Siegel's soothing syrup.

So far these symptoms only point to hyperchlorhydria, which may be present alone or may accompany ulceration.

These symptoms may be present for some time without any further sign, and the patient may content himself with the relief which he obtains in this way.

Should he present himself for examination, however, search should be made for painful pressure points in the epigastrium, or if the patient is a woman and has been in the habit of wearing tight corsets, these pressure points must be sought for lower down, even as low as the umbilicus.

Here, as in everything else, one has to guard against hysterical simulation. Whilst in true gastric ulcer the pain on pressure is only felt over a small circumscribed area and always at the same point in the same patient, in hysteria the discomfort complained of will be all over the gastric area, and in some cases the stomach may be mapped out for the examiner by the patient's sensations. Moreover, whilst pressure increases pain in ulcer, the hysterical pain is relieved by it.

In true gastric ulcer the pain is always in the same spot; when once found, therefore, its exact location should be noted, and this area should be tested several times during successive days. The pain, moreover, being due to the contact of the acid gastric juice with the raw surface of the ulcer, will be most intense whilst that juice is active; it will, therefore, be at its worst soon after that food is taken, and will gradually become less severe as the juice combines with the proteids; it does not, however, disappear until the stomach is emptied, either by vomiting, or more naturally, by the passage of the food onwards into the small intestine.

In some cases a similar spot may be detected behind, just outside and to the left of the tenth, eleventh, or twelfth dorsal vertebræ; but this is not essential for diagnosis, being often absent; indeed, its presence would appear to be more dependent upon the position of the ulcer on the posterior wall than on its mere existence.

When matters are at this stage there may be some hæmatemesis, but the amount of blood *seen* is not generally large in quantity nor is it by any means universal. Brinton says that it occurs in 28 per cent. of all cases, Gerhardt in 47 per cent., whilst both Lebert and Witte put it as happening in one out of every three.

Certainly in some cases, when the ulcerative process has eaten into some large vessel, the amount lost may be great, and the blood may be bright red in colour, but this is very exceptional; the blood usually exudes in small quantities and consequently remains in the stomach sufficiently long for the hæmoglobin to be transformed into hæmatin, and therefore when at last vomited it will have lost the bright red colour of fresh blood, and have gained the fawn colour of coffee. But blood in some amount is almost necessarily present in some form in all cases, and can be detected by appropriate tests.

The large amounts of bright red blood sometimes vomited by chlorotic girls do not usually come from any definite ulcer; in those cases the whole gastric surface seems to exude blood. It is important that this should be remembered, as surgical measures are useless in these cases. Improvement is rather to be sought from remedies which improve the condition of the blood itself, such as lactate of calcium, or those which bring about contraction of the arterioles, as adrenalin or supra-renal extract. It is the small amounts of blood which especially point to gastric ulcer as their source.

But although moral certainty may be said to have been attained by the consideration of these symptoms, the case cannot be considered proven until the actual contents of the stomach have been chemically tested, and because blood is one of the

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most important things for which these tests are applied, and the mere presence of meat would vitiate the results, we apply these tests to the patient at a time when he is fasting and the stomach, if it is not dilated, may be safely supposed to be empty, that is, in the morning after a night's abstinence from food. To such a patient is given a meal which contains no meat, and which experience has shown is capable of exciting the gastric juice as normally as is possible in this condition; one, too, which should be all digested and ready to pass on into the intestine in about an hour*; just before it would so pass on, it is withdrawn by the stomach-tube, and the amount of digestion which has taken place is noted; this is best seen by placing the stomach contents so drawn off in a conical glass for a few minutes, when those contents will separate into three layers; the upper one will be frothy, containing certain gases and air; the lower will contain any fragments of undigested food, such as bits of bread or toast; between these will be the main products of the digestive process.

I will ask Dr. Porter to pass the stomach-tube on this patient in order to show how this manœuvre should be carried out.

You will notice that no elaborate preparation is made, which, often by frightening the patient, excites a nervous spasm of the pharynx, and so defeats the object of the examiner. The tube is simply dipped in warm water, and as soon as the tip of it reaches the back of the tongue the patient's head is brought forward, and the tube glides on and down the œsophagus by the simple effort of swallowing, assisted possibly by slight pressure by the examiner.

In the earlier attempts at passing the stomach-tube, the instrument was dipped in glycerine and the head was thrown back, in order that the examiner might see in what direction the tube was passing; the taste of the glycerine directed the examinee's attention to his throat, and thus sometimes excited the same pharyngeal spasm which it is desired to avoid. If the head is thrown back, the epiglottis is raised and there is a greater possibility that the tube may enter the trachea—the one thing to be avoided; besides, the tube might hitch against its projection behind the tongue, and so set up hawking; by bringing the head forward, although the examiner no longer can see himself the point of the tube, the greater ease with which it passes by the side of the trachea more than compensates for this loss of vision.

The end of the stomach-tube being within the stomach, which is known by the coincidence of this line marked on the tube with the line of the teeth—this line is 38 cm. from the tip—its open end is attached to a bottle through the stopper of which two tubes pass. To the second glass tube a rubber tube and exhaust bottle are attached, by means of which the air in the glass bottle is removed; as this is done, the fluid contents of the stomach rise in the stomach-tube, and flow into the receptacle. This is Ewald's method, and is at once the most simple and the most cleanly.

The fluid so obtained is then strained through muslin, not through filtering paper, which is not easily penetrated by the mucus always present in this fluid.

Five things have to be learnt from this strained material:

First, the total amount of acidity.

Second, the relative amount of free HCl.

Third, the digestive power of the fluid, *i. e.* the presence of pepsin.

Fourth, the presence or absence of lactic acid.

Fifth, the presence or absence of "occult" blood.

*The total amount of acidity per* 100 *c.c. of strained gastric contents.*—Ten c.c. of this fluid is taken and placed in a small porcelain cup; porcelain or white pot is used, because the change of colour is best seen against a white background. To the fluid one drop of phenolphthaleine solution is added and well mixed. This reagent is colourless in an acid fluid, whether that acidity is due to free acid or to an acid combined with proteid, or to an acid salt, but shows a pink colour as soon as all acid is neutralised. The cup is then placed under a burette containing deci-normal solution of soda; drop by drop this solution of soda is allowed to fall into the fluid until the pink colour does not deepen on the addition of a further drop. When that happens, the amount of solution used is read, and the number of c.c. is multiplied by ten, since only 10 c.c. has been tested; this gives the total acidity; thus in this case 5 c.c. have escaped from the burette, which multiplied by ten equals 50 per cent.

* Such a meal is Ewald's test breakfast, consisting of 18 oz. of bread and 10 oz. of weak tea without sugar.

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*The relative amount of free HCl.*—Another 10 c.c. of the strained vomit is placed in another porcelain cup, and 2 drops of amido-azo-benzol is added. This retains its colour only in the presence of free mineral acid; so that this process is practically the reverse of the other, except that the presence of acid salts or acid combined with proteid has no effect one way or the other. The fluid is again neutralised with the deci-normal solution of soda, and the amount necessary to remove the colour previously present is again noted, which in this case is $2\frac{7}{10}$ths, and is multiplied by 10. This gives the relative amount of free acid; and as we know that no other mineral acid can be present, it is evident that it is a measure of the free HCl. In this case, then, it is 27.

*The digestive power of the fluid, i. e. the presence of pepsin.*—Small discs of boiled white of egg are used for this test.

Five c.c. of strained stomach contents are placed in each of three test tubes; in each one disc of egg is dropped. All of them are placed in a water bath heated to body temperature. Nothing is added to the first tube; if the disc is dissolved in one hour, the gastric juice contains enough acid and active pepsin. To the second 2 grains of dried pepsin are added; if the disc is not dissolved in the first tube in an hour, but is in the second, the original fluid was lacking in pepsin. To the third tube 2 drops of dilute hydrochloric acid are added; if in neither of the first two tubes has the disc been dissolved, but this occurs in the third, pepsin was active, but the amount of hydrocloric acid was deficient. If in none of the three first tubes is the contained disc dissolved in an hour at body temperature, the fluid is lacking in both pepsin and free HCl.

This has already been tested in this case, and the disc was dissolved in the first tube in one hour, so proving the presence of sufficient active pepsin and hydrochloric acid.

*The presence or absence of lactic acid.*—To determine this question Uffelmann's reagent is used. This should be freshly made on each occasion as the colour is lost by keeping. It consists of a 1 per cent. solution of carbolic acid in water, to 10 c.c. of which 2 drops of Tr. ferri perchlor. are added, and it has an amethyst blue colour. A few drops of the strained stomach content is added to this solution; if any lactic acid is present the blue colour disappears, to be replaced by canary yellow. In this case no change of colour is seen, therefore no lactic acid is present.

It is to be noted that one of the strongest evidences at present available of the existence of cancer of the stomach is the co-existence of a low amount of free HCl with a detectable amount of lactic acid.

And as all bread contains a slight quantity of lactic acid, if this test is needed, because there is a suspicion of the presence of cancer, the patient should receive, in place of the ordinary Ewald's breakfast, Boas "oatmeal soup," which consists of a teaspoonful of rolled oats boiled in 1000 c.c. of water down to 500 c.c., to which a little milk is added but no salt; in this way no fresh lactic acid is introduced; any found must have been originally present in the stomach.

Salomon's test for cancer is based upon the fact that such growths exude an albuminous fluid; the stomach is washed out repeatedly with saline solution, and the washings, after all food has been removed, are tested for albumen. If any is present in the later amounts this constitutes a strong presumption in favour of cancer.

*The presence or absence of "occult" blood.*—To 5 c.c. of gastric contents add 3 c.c. of glacial acetic acid; shake the two together thoroughly; this transforms any hæmoglobin present into acid hæmatin. To this add 5 c.c. of ether; tilt this from side to side; the ether becomes coloured with the hæmatin which it extracts.

Draw off with a pipette 2 c.c. of this supernatant etherial extract of hæmatin, and place in a test tube; to it add 2 c.c. hydrogen peroxide and 2 c.c. of an alcoholic saturated solution of benzidene, which must be fresh. At the line of juncture a blue or bluish-green colour is seen if blood is present.

As this patient has had gastro-enterostomy performed last November for a gastric ulcer I shall be surprised if any blood is now found, and should think the reaction, if present, due rather to the last meal of meat he may have taken than to any personal loss.

Inasmuch as the most frequent site for a gastric ulcer is the pyloric region, it is evident that in the process of healing contraction of the pyloric orifice is very likely to occur, and as a matter of fact it

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very often does happen that if the ulcer heals after medical treatment combined with rest, difficulty is afterwards experienced in the due emptying of the stomach.

As this difficulty increases the same results follow here as are seen elsewhere when a cavity containing fluid strives to empty itself through a strictured tube; greatly increased and spasmodic contractions of the cavity are at first excited, and should they be capable of overcoming the difficulty some hypertrophy of the muscular wall will result. If, however, the difficulty becomes too great these muscular walls appear after a short period to recognise the futility of their efforts, they gradually yield, and the cavity they enclose becomes enlarged; the muscular fibres themselves undergo fatty degeneration and atrophy—the stomach is said to be losing or to have lost its *motility*.

It is important, if surgical interference is to be of any marked utility, that the operation should not be left until these latter changes are advanced; it is doubtful whether a stomach which has once entirely lost its motility ever regains it, and certainly the improvement which gastro-enterostomy is capable of producing is much more decided if the operation is done upon a stomach which shows peristaltic waves.

Both during the progress of a gastric ulcer and later, when the ulcer having healed, stenosis of the pylorus is produced by its contraction, the operation of gastro-enterostomy has proved to be the most effectual treatment.

During ulceration the irritation set up by the presence of the ulcer excites increased activity of the acid-forming glands, and both together cause spasm of the pylorus, so that the hyper-acid contents of the stomach are kept for a longer period than normal in contact with the ulcer in its walls. The otherwise possible healing of this ulcer is thereby greatly delayed, if not prevented. Fibisch produced gastric ulcers artificially in dogs, and demonstrated that after they had existed for some time without showing the least tendency to heal, when gastro-enterostomy had been performed they had healed in a few days. By permitting an earlier exit through an opening not controlled, as the pylorus is, by a muscular sphincter, this extremely irritating fluid is prevented from such prolonged action upon the stomach wall. In addition, Patterson has shown that after such an opening has been made, some of the alkaline contents of the small intestine enter the stomach and thus tend to neutralise the excessive acidity. More important still, Pawlow demonstrated that the presence of an acid fluid in the jejunum reflexly excited the secretion of pancreatic juice, and that the appearance of this put a check upon the secretion of the gastric secretion, so that less acid was poured out, and what was present was sooner neutralised. The natural fear that food might escape too soon, and not having been sufficiently acted upon by the pepsin and acid in the stomach might fail to be rendered fit for absorption by the lacteals, or might be absorbed in an unfit state for general utilisation, has been by experience proved to be groundless.

Certain observations through the fluorescent screen by Dr. Barclay and myself have shown that after an opening between the stomach and jejunum has been made farinaceous food escapes at once, but that proteid foods are still retained for a time before passing onwards, the opening being apparently closed by the contraction of the muscular wall around, which later yields and allows its passage, whilst many experiences of the operation itself have shown that nutrition, instead of being impaired, is greatly improved when once this has been done.

And in the after-stage of gastric dilatation great good has been effected. Food too long retained in the atonised bag produced at the fundus tends to ferment and even to decompose if not digested. Gases of various kinds are formed, which still further dilate the weakened walls of the stomach; toxins produced there are absorbed into the general circulation and poison the unfortunate possessor of a dilated stomach. All this is put an end to by the operation if not too long delayed. In cases in which there has been delay, and consequently but little power of recuperation exists, it is often useful after the first fortnight, when the union may be considered sound, to resort once more to gastric lavage every third or fourth day for a time. One cannot expect a much dilated stomach to return at once to the normal size, although Kreuzer, who examined Kronlein's patients, says that it does ultimately regain its original cubic content. In one of our patients within three weeks great improvement was seen in this direction. In another no particular progress appeared to be made after six

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weeks until lavage was again instituted, after which the size of the stomach fairly rapidly became less, though it never quite regained, during the six months she was under observation, its original size. This patient was æt. 54 years, and had complained of stomach troubles for six or seven years. Before operation her stomach was extremely dilated, its lower border reaching the symphysis pubis, and when dilated with carbonic gas filling the entire abdomen.

Post-operative gastric lavage undoubtedly appears to be useful in keeping the gastric mucosa clean, and preventing any possible retention until the muscular wall has recovered its tone.

There are difficulties in obtaining accurate data for comparison of the size of a stomach before and after operation. In order to estimate the full amount of dilatation the patient must be erect; in the dorsal position of the body the fundus of the stomach occupies a much higher relative area in the abdominal cavity; as after operation but few patients during the first ten to fourteen days are fit to be radiographed in the erect attitude, the apparent improvement as to position may be very misleading, being simply due to the altered posture. It is therefore only by comparing the difference thus produced before operation and allowing for it afterwards that any reliable estimate can be made; and this must be done in each case, since posture appears to produce a variable effect in different subjects.

We are engaged at present in such an investigation and its results promise to be interesting, but our work is not sufficiently advanced to warrant as yet any definite statement.

In order to obtain the best results from operation the method employed is of great importance. In the older methods, as further experience has shown, many unnecessary complications were introduced. The jejunum was attached to the front of the stomach; this necessitated a long loop of gut, and this loop had to pass over the transverse colon; if the loop were short there was a danger of constriction of the large bowel by the mesentery of the small and of tension upon the jejunal mesentery, thus narrowing the calibre of its vessels and imperilling the nutrition of the attached gut. If the loop were a long one, whilst these dangers were avoided, the contents of the duodenum and upper jejunum tended to accumulate in the afferent limb, which became distended. It was practically almost impossible to prevent some angulation of the loop at its gastric attachment and this further delayed the exit of the contents of the afferent limb; the weight of this distended gut pulled upon the attachment and thus in its turn increased the tendency to the formation of an angle. Each element in the difficulty reacted upon the other and increased it; when at last the distended afferent limb contracted upon its contents and forced them onwards, so large an amount was suddenly carried past the artificial opening that had been made that a quantity far beyond what that organ could tolerate overflowed into the stomach; this untoward event was, of course, greatly increased by the probability of any angle at the point of union. A case illustrating this is reported by Dr. King, of Cambridge, in the 'Lancet,' October, 1907; vomiting, which was copious and persistent, followed the operation; seven days later the abdomen was again opened and the afferent limb nearly two feet in length was found distended and dilated whilst the efferent was collapsed.

When these operations were first done speed was often the most essential element in success; the patients were not submitted to surgical treatment until they were exhausted, emaciated, and in great danger; the most rapid method was found in the use of a very ingenious appliance invented by Dr. Murphy, of Chicago, and known as Murphy's button. This, however, whilst it supplied the means for a swift operation, introduced a foreign body which had later to be got rid of; numerous instances of obstruction produced by its presence were seen; at present the patients come to the surgeon at an earlier period; speed has ceased to be of so much importance and the best method can be carefully and deliberately carried out without fear of collapse during the work.

By a process of gradual evolution and with the collective experience of many operators, the present posterior method has been evolved. Von Hacker was the first to carry out the posterior method, but many additions have been made to his original suggestion. It has been proved that it is not necessary that the gut and the stomach should be iso-peristaltic so no loop at all is now formed, but the upper part of the jejunum is simply attached just beyond the duodeno-jejunal angle to the posterior gastric wall, reached through an avascular

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space in the meso-colon, in such a way that the distal end of the gut lies lowest. The opening must be a free one, from two and a half to three inches long; many experiences have shown that if this is not done, not only is there a much greater risk of re-closure, but that even whilst the opening is patent the best results are not obtained. The method of suturing should ensure that no raw surfaces are left, but that the mucosa of one cavity is continuous with that of the other. There should be no foreign body present, acting as a support or otherwise. They are not necessary, and may, as already pointed out, introduce additional and serious risks.

The after-treatment of these cases is also important. If the operation has been done for ulceration, one can scarcely place the patient too soon in the Fowler position, *i.e.* almost sitting upright in bed. In most cases for the first few hours it is necessary to allow the dorsal position until the post-operative exhaustion has passed away, but as soon as this is over the sitting position should be assumed, since in these cases one wishes to drain as soon as possible, and the stomach, not being proptosed or dilated, the artificial opening will be most effective with the body erect.

But that is not so if the operation has been done for dilatation. Here, as our observations show, the stomach fundus sags downwards far more in the erect than in the flat dorsal position; there is greater danger that food may accumulate at a lower level than that of the opening provided; besides, the proptosed stomach will drag upon the attached jejunum, and this attachment being close to the one relatively fixed point, the duodeno-jejunal angle, will tend to drag this lower still, besides narrowing the calibre of the intestinal tube at this point by the traction so exerted. As we have seen, the gastric fundus lies at a higher level in the abdominal cavity when the patient is supine, and therefore less or no drag will be exerted upon the small intestine united to it. Experience has shown that this reasoning is approximately correct; discomfort experienced when such a patient is placed in the Fowler position disappears when he is allowed to lie flat in bed, whilst the stomach appears to recover sooner its normal tone.

Post-operative vomiting is very rare, beyond the ordinary sickness following an anæsthetic if the later technique is followed at the time of operation, and gastric lavage is seldom required in cases of ulceration; indeed, it should generally be carefully avoided. In dilatation, on the contrary, as already mentioned, it sometimes appears to be very useful after the first fourteen days; it should never be attempted earlier.

Great difference of opinion exists as to the question whether gastro-enterostomy produces drainage of the stomach or not. The observations of Dr. Barclay and myself seem to show that in the earlier days after operation at least, and with farinaceous food, it does so act, whatever may be the case later; this is the more important, because the feeding soon after the surgical interference is mainly farinaceous; it is only later that proteid foods are given. In one case, examined some days after operation, the farinaceous food passed through the stomach completely and was seen in the intestine within three minutes; four weeks later, part passed at once, and part remained, the opening closing for ten minutes, and then permitting the remainder to escape.

In cases of ulceration I have found the most suitable initial food to be cream with soda-water; this forms a very palatable mixture, and is perfectly unirritating. Many patients find that it allays the irritability of a stomach recovering from an anæsthetic. And when it is remembered that oil is the best remedy for use in gastrorrhea, and the sedative action of soda on the irritated gastric wall in gastritis, the scientific value of such a combination appears evident. At any rate, in practice it is found to be very effective.

Useful as it is in ulceration, however, it is not of much value in dilated stomachs; in these cases Benger's or Mellin's food made with water and given in small quantities appears to be the most suitable at first; to these milk is gradually added and the amounts are increased with fair rapidity. Later, other foods are added to the dietary.

*September 28th*, 1908.

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We have received from Messrs. Allen & Hanbury a copy of their new and greatly enlarged price list. The first section embraces drugs, chemicals, and pharmaceutical preparations. The third section will attract special attention because of its subject being serum and vaccines; it contains an interesting description of the technique employed in determining the opsonic index. We hope to find space in a future issue for a fuller notice of this useful publication.

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# EARLY CLINICAL TYPES OF CERTAIN DISEASES.

Clinical Lecture delivered at University College Hospital.

By WILFRED TROTTER, M.S., F.R.C.S.,
Surgeon, East London Hospital for Children; Assistant Surgeon, University College Hospital.

THERE is no matter upon which the agreement of surgeons is more complete than it is in regard to the importance of early diagnosis. In the present state of surgical therapeutics it cannot be denied that for the most part the advanced stages of the more serious diseases are incurable, so that satisfactory treatment of them is directly dependent upon early diagnosis. Unfortunately the body possesses a great capacity for tolerating without disturbance the early stages of many diseases of the gravest kind. The earlier symptoms may be such as scarcely to attract the attention of the patient, and even if they do there may not be signs enough to allow of the surgeon making a definite diagnosis, or making a diagnosis at all or even suspecting that there is anything serious the matter. The natural unavoidable difficulties then are serious; but it is a fact which cannot be overlooked that there are certain defects of diagnostic method which add unnecessarily to the difficulties of these problems. We may fail to make a sufficiently extensive or thorough examination; we may be too easily satisfied that we have discovered an adequate cause of the symptoms, we may not be sufficiently skilled in examination to detect important signs, or have sufficient knowledge correctly to interpret them. There is, however, one very important cause of errors and delays which I propose especially to deal with to-day, and that is the failure to grasp what is the minimal body of symptoms and signs upon which an adequate diagnosis can be based. Thus it comes about that time is lost in unnecessary waiting for diagnostic developments in a case which is either already clear or in such a state as to demand exploratory operation. It is therefore of fundamental importance in learning the symptoms of a disease to get a firm grasp of what constitutes the smallest number of symptoms upon which a diagnosis can be based. Such clinical pictures of minimal groups of symptoms must unfortunately be constructed more or less independently of the text-books. It is remarkable how little those increases in our knowledge of early stages of disease, which the great extensions of the possibilities of operative surgery have given us, have been assimilated with current pictures of disease. Many such pictures date from a time when diagnosis could be verified only in the post-mortem room, and when, therefore, the later aspects of disease tended to become fixed as the ordinary types. I propose to deal to-day with four conditions, of which it can be fairly said that the accepted clinical accounts present aspects of the diseases which are distinctly late and for the most part either unfavourable for treatment or actually incurable.

*Carcinoma of the breast.*—Let us begin with a thoroughly common condition, such as carcinoma of the breast. What are the signs which are generally accepted as constituting the ordinary picture of this disease? A hard nodular mass in the breast fixed to the breast substance, causing retraction of the nipple and dimpling of the skin and attached to the pectoralis—such might pass as what is accepted as, at any rate, a fairly early case—and we might add the presence of enlarged glands in the axilla. Now, the point I wish to bring before you is that such a condition is an advanced one, and that if such a description continues to be a generally accepted one, we shall go on having to deal with advanced cases with the familiar depressing results.

Now, when we examine this description we find that it embodies many features in excess of those which are necessary for the arousing of a suspicion of carcinoma sufficiently strong to justify operation. What, then, we may ask, are the minimal signs upon which can be based a judgment sufficiently certain to render an operation necessary? As always, rational diagnosis must be based here on morbid anatomy and pathology, so that to get such help as we are in search of we must examine into the characters of specimens of carcinoma of the breast, asking ourselves what are the signs which are essential, and must be present in all cases. Now, if you look at a specimen of the common scirrhus of the breast you will find that the cancer lies in the breast substance, of which in fact it actually is a part, that it is much harder than the breast, so that when the tumour cannot be detected with the

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eye it can be felt by the finger, even if the gland substance is fibrous, and therefore unduly firm. This characteristic causes the tumour to be palpable as a distinct well-defined lump in spite of its being fixed to the breast substance. Finally, the tumour is solid. Now none of these characters will be affected by the earliness of the stage of the disease. If a scirrhus of the breast can be made out at all, however small it is, it must have these characters. Therefore, any condition of the breast presenting these features must arouse a strong suspicion of carcinoma, and, as in case of carcinoma early treatment is essential, such conditions demand exploratory operation with absolute urgency. It therefore may be laid down as a rule, with no exception, that a *hard, solid, well-defined mass fixed to the breast substance* must be explored, for it is practically certain to be a scirrhus. The question may now be asked, if we accept this rule, What risk is there of finding at the operation some other condition?

In the first place we may make a mistake in the observation of the physical signs. The hardness, definition and fixation are not very likely to be mistaken, but the other sign, namely the evidence of solidity, is one which is sometimes extremely difficult to make out. There is only one group of conditions having all the signs of early scirrhus except solidity, and that is a collection of fluid surrounded by a thick wall such as a chronic abscess or a cyst embedded in a mass of chronic inflammatory tissue. In both cases, however, the disease must be dealt with by operation, so that in advising exploration the treatment will have been the best possible.

There is one other condition which sometimes very closely resembles an early scirrhus, and that is gumma. This may appear as a hard solid mass fixed to the breast substance. It is, however, much less common than scirrhus; careful examination will sometimes show that it is not actually in the breast substance; its outline, as is almost always the case with gummata, is generally less distinct than that of a carcinoma on account of the characteristic diffuse surrounding fibrosis. Moreover, gummata rarely occur singly, so that there will usually be evidence of present or past gummata elsewhere.

Thus, if we accept the rule of regarding all hard, solid, fixed lumps in the breast as scirrhus, we may occasionally encounter at the exploratory operation conditions such as chronic abscess or cyst, which, it must be borne in mind, are themselves indications for operation, or we may very rarely, if we forget to consider the possibility of its very exceptional occurrence, explore a gumma; but we shall save a large number of cases of cancer of the breast from being left for treatment to a stage when, instead of the outlook being good, it is almost as black as it can be.

*Traumatic compression of the brain.*—Let us now pass on to another example. Compression of the brain by a traumatic hæmorrhage is usually described as being characterised clinically by coma, stertorous respiration, slow pulse, high blood-pressure, dilatation of the pupils, and perhaps hemiplegia. Now I want to point out to you that this which masquerades as a description of ordinary cerebral compression is in fact a description of the last stages of the condition, the state from which the possibility of recovery is already extremely doubtful, the state, in fact, which only just precedes the onset of the evidences of that paralysis of the bulbar centres, which is the immediate precursor of death. The attitude towards cerebral compression, which regards the picture I have mentioned as a relatively early stage, is only possible if the essential features of the pathology of the condition are ignored.

The feature in the pathology of compression which it is of absolutely cardinal importance to recognise is that when the condition is due to a hæmorrhage—such, for example, as one from the middle meningeal artery—it is, without exception, primarily local, and will produce, therefore, its most marked effects on the brain in the immediate neighbourhood of the clot, while the more distant parts will be involved proportionally to their distance, and, as the condition increases gradually, proportionally to the time which has elapsed since the onset.

The symptoms produced by the compression are most conveniently grouped into those due to the effects on the hemispheres, those due to the effects upon the mid-brain, and those due to effects on the bulb. The first group will include impairment of consciousness and motor changes, the second will include pupillary changes, and the third changes in the respiration, the action of the heart, and the

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blood-pressure. In each group there will be two stages of the symptoms—an early irritative and a late paralytic. Thus, among the hemispheral symptoms, we shall have at first restlessness, delirium, rigidity, and convulsions, and later drowsiness, coma, paralysis, and changes in the deep reflexes. In the second group, at first contraction of the pupil, and later dilatation. In the third group, at first slow, strong pulse, stertorous respiration, and high blood-pressure; later irregular, gasping breathing, small, rapid pulse, and a rapidly falling blood-pressure. We thus have six groups of symptoms in all, and as, in the majority of instances of cerebral compression, the clot is over the anterior part of the hemisphere, these symptoms will appear roughly in the order in which they have been given, because the effects of pressure will be felt first in the hemisphere, then in the mid-brain, and only relatively late in the bulb. It is quite clear, therefore, that the classical picture of compression which is currently accepted as the minimum adequate for diagnosis represents a very advanced state of the condition because it contains as essential features symptoms of definite involvement of the three great vital centres of the bulb. What, then, we may ask, is the minimal group of symptoms which is adequate for the diagnosis of compression in an early stage? To answer this question we must consider briefly the mechanism by which the symptoms are produced. In the first place, none of the symptoms are due to the direct effect of the clot upon the part of the brain concerned, but all are produced indirectly through the effects of the pressure upon the circulation in the brain. Obviously the first effect of such compression will be to obliterate the veins and produce venous congestion, and this venous congestion it is which produces the early type of symptoms, all of which are irritative in character. Increase of the pressure upon the brain must press the blood out of the capillaries and so cause anæmia. This anæmia causes the second type of symptoms, which are all of them paralytic. Now it is a matter of experience that states of compression affecting the hemispheres severe enough to cause widespread venous congestion and the consequent irritative symptoms are recoverable from without operation, as is shown by the fact that the cases of so-called cerebral irritation, which are clearly nothing but widespread slight compression, usually recover without interference. As soon, however, as the compression is sufficiently great to produce anæmia, and therefore paralysis of however slight a grade and small a distribution, we know that we are dealing with a condition which in the great majority of cases is not recoverable from without operation. We may therefore lay it down as a rule that in a case of head injury the appearance of definite evidence of paralysis, whether it be a hemiplegia or a monoplegia, a unilateral elevation of temperature or a unilateral change in the deep reflexes, is an absolute indication for operation. This applies to symptoms of the mid-brain group as well as to those of the hemispheral group, but it must be extended when we deal with compression in the posterior fossa. The functions of the bulb are so important that the onset of anæmia of the bulb, and therefore of paralytic symptoms, means almost inevitable death. The practical consequence, therefore, is that irritative bulbar symptoms must be accepted as an indication for operation as absolute as are paralytic hemispheral symptoms.

*Malignant disease of the thyroid.*—Yet another example of the importance of the principle which I am trying to expound is to be found in malignant disease of the thyroid. This is a condition which, to the shame of surgery, is practically invariably fatal, not because it is from the onset incurable—for we have enough experience to know that it can be cured—but because of the very widespread belief that the diagnosis must be based on the presence of a hard, nodular, fixed mass involving the recurrent laryngeal and perhaps the sympathetic nerve. Indeed, you may actually find it stated by many authoritative writers as a sign of the malignancy of a goitre that the tumour surrounds instead of pushing aside the carotid sheath. Such a "sign" would be better called a death warrant. There are, in fact, however, evidences produced by malignant disease of the thyroid while yet in an operable condition, which indicate the presence of the condition with sufficient certainty to make operation imperative. Carcinoma of the thyroid never appears except in a gland previously abnormal, and when it does appear produces changes similar to those found in scirrhus of the breast. This minimal picture can be stated in a very compact form. A patient who has had a goitre for some years notices that it is beginning to increase in size

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and is becoming harder. Physical examination reveals a swelling—it may be quite small—which has no longer the perfectly distinct and globular outline of an adenoma and is not movable within the substance of the thyroid. It is hard, nodular, and can be felt to be welded with the thyroid substance. Such a tumour must be operated on, though there is no limitation in the movement of the thyroid and trachea during swallowing, no compression of the œsophagus, and no involvement of the trachea, recurrent laryngeal nerve, or carotid sheath.

*Actinomycosis of the lung.*—A last example will be taken in a disease which is almost universally regarded as incurable in any stage, namely, actinomycosis of the lung. It is a significant fact, however, that the cases which reach the surgeon do so for the most part in the later stages, for the disease usually goes unrecognised until it produces manifestations which refuse to allow themselves to be overlooked. We then have the fairly familiar picture of the patient with profound wasting, a hectic temperature, marked dyspnœa and perhaps cyanosis, and a tumour of the chest-wall, and probably enlargement of the liver. In order to obtain such a conception of the disease as will enable one to suspect its existence before the patient is moribund it is necessary to get some idea of its pathological anatomy. Actinomycosis, like tubercle, is a granuloma of the lung which begins as a chronic broncho-pneumonia. In its development, however, it differs from tubercle in two important particulars. It tends to infiltrate the tissues regardless of all natural boundaries, and it does not break down into cavities of any considerable size. The practical corollary of the first characteristic is that it involves the pleura early and tends to cause widespread adhesions (a matter of great importance in treatment) as well as throwing large tracts of the lung out of action. A consequence of the second characteristic is that the evidences of destruction of the lung are apt to be very slight. Early actinomycosis, then, is a disease of symptoms rather than of physical signs; its clinical appearances may shortly be described as those of a basal phthisis producing marked shortness of breath, no profuse hæmoptysis, and few or no signs of cavitation, with a sputum which contains no tubercle bacilli and no elastic fibres. Such a picture, then, is fairly characteristic and, at any rate, should give rise to a suspicion sufficiently strong to encourage us to examine the sputum for the specific fungus. If a diagnosis of the disease can be made at this early stage there is reason to suppose that energetic operative treatment would be successful, and would give us a chance, at any rate, of removing from surgery the painful reproach of one of those diseases the diagnosis of which is a death sentence.

*September 28th*, 1908.

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**Bacteriological Examination of the Blood of the Sinus in the Differential Diagnosis of Otitic Sinus-thrombosis.**—Nuernberg ('Munch. med. Wochens., Jahrg. liv, No. 51) says that the diagnosis of otitic sinus-thrombosis rests up to this time almost entirely upon the temperature rise. Some cases, however, in which there is for some reason no rise in temperature are therefore not diagnosed; also some cases of otitis accompanied by high fever are not examples of sinus-thrombosis. The method consists in taking at the same time blood from the sinus and from a vein of the arm and making cultures of it. In the event that the number of colonies of bacteria in the culture from the sinus greatly exceeds the number in the blood from the arm vein, then it is to be concluded that the thrombus lies much nearer the point of puncture of the sinus than the point of puncture in the arm, for in thrombosis, when the blood retains its bactericidal properties, the farther removed from the seat of the thrombus the fewer the bacteria. Of the cases investigated in this way there were four of sinus-thrombosis in which the sinus blood showed numerous colonies of streptococci in pure culture, while cultures from the arm vein were sterile. In a fifth case of beginning sinus-thrombosis the cultures from the sinus blood also remained sterile. In the pre-thrombosis stage bacteria are also present in the sinus blood, therefore bacterial examination alone is not sufficient to settle the diagnosis when the cultures are positive. In such event the sinus walls should be exposed, and there should then be a delay of three days to see if the fever subsides or not. If it does not, sinus-thrombosis is present. Many more experiments are necessary in order to establish the correctness of this means of diagnosis.—*The Therapeutic Gazette*, vol. xxiv, No. 8.

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## AFTER-EFFECTS OF INJURIES.*


By CLINTON T. DENT, F.R.C.S.,
Senior Surgeon, St. George's Hospital.


I HAVE had during the last few years some exceptional opportunities of studying the after-effects of various injuries particularly in respect to the wage-earning capacity of the individual. The importance of the after-results and after-effects, the degree to which recovery can take place, is of course a matter of much greater moment to the wage-earning class than to those who are in easy circumstances of life. No doubt in the near future the whole question will demand still more attention owing to the trend of opinion and recent legislation. There is little published information on the subject. The experience of those who have to deal with large bodies of men, who, from the nature of their employment, are exceptionally liable to injuries is not readily accessible. For the most part the cases are soon lost sight of, being dealt with summarily, and so the real after-effects are almost unknown. Occasionally endeavours have been made to follow up hospital cases, but so far as I am aware, diseases rather than injuries have chiefly attracted attention. Moreover, hospital patients are not good correspondents. No great reliance can be placed on such replies as are received, and it is but seldom that the all-important personal observation can be exercised. Still, the class from which hospital patients are drawn, or perhaps it would be better to say, should be drawn, is precisely the one in which after-results are of prime consequence, not only to the individual but also to the community.

My own experience is largely derived from a picked body of men—the Metropolitan police. These men are drawn from all parts of the United

* Delivered before the Medical Society of London.

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Kingdom. As might be supposed a large proportion come from the country districts. Every effort is made to secure sound, healthy men at the outset. A certain leaven of men who have served in the navy and army is accepted, but in these cases full details as to the previous medical history are forthcoming. In passing, I may note the deplorably large number of soldiers who are rejected on the score of constitutional syphilis. If a tithe of the efforts that are being made to deal with the scourge of tuberculosis were made to control the spread of syphilis, the gain to the community would be enormous. But while unrelenting war is being waged against the tubercle bacillus the pallid spirochæte is allowed to pursue its devastating course almost unheeded, wrecking and ravaging where it does not destroy. The age for acceptance of candidates is between twenty-one and twenty-seven. Few of the men in the subordinate ranks are more than forty-five. The men, then, are at the outset young, and as vigorous a body as can be secured. They lead a healthy, largely out-of-door life, well housed, fed, clothed, and under close medical supervision. Save that they are more subject to such troubles as are incidental to exposure to the weather, especially when on night duty, there are no disorders to which they are liable that can properly be called occupation diseases. The work, however, to which they are called is highly responsible, and of this responsibility the men are conscious to a much greater degree than might readily be imagined. This is a fact that, as I shall endeavour to show later, has an important bearing on the after-effects of injuries in many cases. The men are enlisted for a term of twenty-five years. If, owing to injuries with which alone I am now concerned, they are invalided out with at least fifteen years' service, they come on the pension list; and if the injuries are received in the execution of their duty they are pensioned from the date of invaliding, however short their service may have been. But at any time up to twenty-five years after enlistment they can be required, if fit, to rejoin, or forfeit their pension. All these pensioners I am able to watch closely, seeing the men at intervals varying from three or six months to three years until the term of their liability for service has expired. Thus the effects of injuries can be studied often over long periods. The bulk of the men are actually seen, but in a proportion of cases a medical certificate supplies the necessary information. Probably there are not many equally good opportunities of estimating the after-results of injuries, for it is always possible to keep a close watch. The men, it may be noted, are not affected by the Employers' Liability Act or similar legislative acts.

Medical officers connected with large factories or those who practise in the mining districts must have a very large experience of accidents and injuries of all kinds, but if the sufferers are incapacitated from work thereby not much more is heard of them, and if they are able in due course to resume their former employment the after-effects are not likely to be systematically studied. Moreover, the standard of capacity to resume work is very much lower in these instances. It matters comparatively little to a man engaged in purely manual labour whether, for example, slight lameness persists after an injury to the lower limbs, or whether there is, say, some impairment of his special senses after a head injury. The great factor of responsibility in connection with the occupation is usually wanting. Those who have to deal with bodies of men, such as railway servants, have a more analogous class to deal with, and their experience would be of great value, but it is not easily to be obtained on any large scale. In the case of railway servants, too, there is more diversity of work. A man, *e.g.*, who has met with a head injury which might render him unfit to follow responsible duty, such as that of a pointsman, can be shifted to employment which makes less demand on his reliability and nerve, but in the case of police, unless an officer has special training and abilities fitting him for clerical or similar work, there is no variety of occupation. There is practically no light duty which can be assigned to him. He has to do the full ordinary work or none. Highly coveted special appointments cannot be filled up by invalids. Cases of injury in connection with railway accidents are very comparable in many respects to some of those to be considered directly. They come up frequently enough in the Law Courts, and the after-effects that may be anticipated form the chief matter of argument, but here the matter is settled when the damages are once determined. The actual remote effects are not invariably in accordance with the prognosis given by the expert witnesses, and in any case the patient's after-

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history is not followed up or known. The perusal of medical evidence given in actions at law where injuries are in question does not, as a rule, furnish instruction of any marked value. A medical witness is apt, on such occasions, to become too much of an advocate and the lawyer too much of a doctor. Neither of the parties is particularly well fitted for the *rôle* for which he casts himself.

It is far from my intention under the guise of an "oration" to embark on anything in the shape of a statistical paper or with the mere recital of cases. I wish rather to set forth some general conclusions based upon the experience described and to allude only to a few special instances in support of those conclusions, and will endeavour to deal with these mainly as they concern civil and particularly hospital practice.

Injuries to police, as might be imagined, are of very frequent occurrence. The only figures that need be quoted are mentioned merely in order to illustrate the risks that these men run. The degree of risk has a great bearing on the after-effect. The injury may be recovered from almost wholly, or absolutely. Broken bones may unite or a wound heal perfectly; but from the mental effect a man does not recover so completely even after a long period of time. The published official report shows that taking a single recent year 2800 injuries are tabulated, so that more than a seventh of the whole force sustained some kind of injury; 2700 of these injuries were due to assaults by prisoners, and in 1750 of these cases the assailants were drunken persons—a possible argument in favour of the Licensing Bill! When it is remembered that these are the figures for a single year it will be seen that an officer's chance of being injured in the course of his career is very great. Indeed, it is the rarest thing to find an officer of any length of service who has never sustained some kind of injury from such causes, and usually, if his lot is cast in the rougher parts of the Metropolis (a very extensive area) there are many such injuries recorded. Of course, the vast majority of these tabulated injuries are of an exceedingly trivial nature from the purely medical point of view, but figures give no real idea of the loss in efficiency, or, in other words, of the mental after-effects.

These after-effects might seem comparable to those that could be gathered from military statistics in times of active service. But any such comparison would be most misleading, for in the case of the soldier the nature of the injuries is usually different, risks are only occasionally run, and the sense of personal responsibility is largely absent. The soldier does not go about his work single-handed; there is much more of finality about any emergency that he is called upon to meet, and to a far less extent has he to thrust himself cold-blooded and on his own initiative into situations where he is likely to meet with injury. Thus the psychological conditions are wholly different. The experience of a soldier who has been with many others in even a serious action differs greatly in its nature from that of a man who finds himself single-handed, as happens over and over again, in the presence of a snarling, hostile crowd, and whose duty requires that he should place himself again under precisely the same conditions at the very first opportunity. As I shall hope to suggest later a point that is far too frequently lost sight of in estimating the after-effects that are likely to accrue from an injury is the conditions under which that injury was sustained.

Allusion need only be made now to a few of the commoner injuries. First of all, with regard to a subject which has occupied a good deal of attention of late in this Society and elsewhere, viz. simple fractures of the bones in the lower extremity. The gravity of these, however simple, when involving both bones is probably not adequately realised by those engaged in hospital practice. Criticism would be out of place on such an occasion as this, as there is practically no opportunity of reply. Still, to those who have facilities for following out these cases the conviction is likely to be borne in that even in the simplest fracture of the leg the period of disability is more prolonged and the after-results are in certain important respects much less favourable than the majority seem to imagine. Investigation on a large scale of the results of simple fractures treated—say in London hospitals—would reveal a very appreciable impairment of the wage-earning capacity. Though by no means true of all, such injuries affect the labouring classes in a great variety of occupations. A new feature has sprung up of late that affects adversely these classes unless their recovery is very complete. The damaged individual frequently finds it hard now to resume his former employment, and harder still to obtain other work. The effect of a fracture of the

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leg might appear to amount to little more than a mere limitation of activity for a measurable period of time. But an employer now is likely to look askance at a working man who is not sound. And no one can really blame the employer if he adopts this attitude. Compensation may seem a great boon at the time, but it is dearly acquired if it permanently stamps the individual, rightly or wrongly, as a damaged article. In the class of men whom I have immediately in view very complete recovery, not far short of a *restitutio ad integrum*, is necessary if they are to resume their occupation. Such a degree of recovery is seldom attained. Slight shortening, or a very trifling degree of limping which might escape notice in other walks of life, would be held to constitute disability.

The fractures that come under observation have been treated by considerable diversity of method. In some, early fixation in plaster or similar apparatus has been adopted; some have been treated by prolonged splinting; others by early massage, and some by immediate open operation. In noting the results and after-effects it is best here to avoid controversial matter, and any discussion as to the respective merits of the various methods would be out of place. At the same time the early fixation in plaster for a period judged to be long enough to allow of sound union while it may give less trouble in treatment, appears to me undoubtedly to delay adequate restoration of function longer than the other methods. Recovery from the point of view of the surgeon and that of the patient does not always coincide in date. The ultimate percentage of disability varies but little whatever method of treatment has been employed. For the most part the fractures that have been treated by open operation may be held to have been probably more severe, and therefore more likely to lead to disability than fractures treated by the older methods of rest and fixation. But the conclusion of the whole matter is that a fracture of the leg in its after-effects is from one, and that a very important, point of view to the individual, viz. his wage-earning capacity, a very serious injury. An injury at any rate that keeps a man from following efficiently a fairly, but not unduly, exacting employment for six months under most favourable circumstances is in itself serious, but if further, as constantly happens, he breaks down when he resumes and is permanently incapable of following that work as a direct consequence of the injury, the after-effects must be regarded as extremely grave. As already mentioned the standard of efficiency is high in the police, the class of men principally under consideration. The work demands fair activity; thus for either two periods of four hours, or for one tour of eight hours, a man must be continually on his legs day after day, though with frequent short spells of leave. Judged by this standard and desirous of keeping well within the mark, my estimate is that at least 30 per cent. fail to attain this standard after simple fracture of both bones of the leg; very few regain it under six months. Practically every case has been treated in the Metropolitan Hospitals, a very few, however, in cottage hospitals and the like institutions within the metropolitan area, which is fifteen miles round Charing Cross. This leads me, with all reserve, to glance at the attitude of hospitals generally with regard to injuries of this nature. The question is a delicate one, even when regarded from the most detached and impartial point of view. It may be noted that hospitals have come more and more of late to be regarded by those who are in medical charge as educational institutions. There is no real reason why this fact should be ignored or suppressed. It is a feature that agitates the minds of some, but yet one that should be encouraged for the simple reason that while essential for the student, it is undoubtedly of great and direct value to the patients. No doubt for the most part accident wards in a hospital are looked upon as of less value for teaching and learning purposes than other departments. Commonplace cases, such as simple fractures, usually excite but little interest. This does not imply that they are in any measure neglected; still, few would deny that patients with simple fractures are hardly as closely supervised by the visiting surgeons as cases of more pronounced clinical value. There is practically, of course, great difficulty in exercising such supervision; sometimes such cases scarcely come within the cognisance of the visiting surgeon. Further, the recently developed tendency in hospitals, deriving assistance from the King's Hospital Fund, to pass as many patients as possible through the wards, while it may be financially advantageous to the institution can hardly be entirely beneficial to the inmates. In face of the

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great pressure that exists in these days on all hospitals, the time and personal attention that would be devoted to a simple fracture in a private case can hardly be accorded to the hospital patient. Such patients necessarily make way early for someone else. This must have some appreciable influence on the after-effects, if only in the direction of prolongation of the time that elapses before recovery up to the requisite point takes place. Fracture of the leg involving the knee or ankle-joints, or fractures of the femur, uniformly lead to permanent unfitness for the work that devolves on these men; fractures occurring in the neighbourhood of these joints nearly always have the same result. I can hardly recall a case of Pott's fracture, or a Dupuytren's fracture where recovery has been complete enough to enable a man to resume police work. Open operation in such cases may effect a vast improvement, but it will not succeed in restoring the patient to the required standard of efficiency, and it is probably not too strong an assertion to maintain that fractures of this nature involving the ankle-joint almost without exception in all the labouring classes lower, even if they do not more gravely interfere with, the wage-earning capacity. My experience with regard to fractures of the leg has led to the expression of rather gloomy, perhaps pessimistic, views.

It is a relief to turn to another class of fractures, viz. those of the patella. Here the results after operation are constantly now all that can be wished, and an injury that was held but a few years ago almost without exception to render a man permanently unfitted for this particular kind of arduous work is now one in which complete restoration of function and strength in the limb can be confidently anticipated. To stray for a moment beyond the confines of the subject in hand, they can be compared in the satisfactory nature of the results to those of operations for hernia. Of the after-results of these a great number are seen, and at periods long subsequent to the operation. Whatever technical method has been employed, they almost without exception justify the term of "radical cure."

I may turn to a less mechanical and more interesting group of cases. After-effects of a psychical nature are frequently determined by the circumstances under which the injuries were sustained. The actual injuries need not have been, in their purely surgical aspect, of any gravity. One or two instances, taken almost at random from a large number, may be cited in support of this assertion. Cases of the kind in hospital practice are likely to pass unnoticed, for it is hardly possible to follow up patients who need only treatment in the Casualty Department.

The following is a typical instance: Some few years ago a young constable in the darkness of a winter evening was called to a house in a fashionable quarter of London, as there was suspected to be a burglar on the premises. The man had apparently got to the back of the house. The constable went in by the side gate and met a man in the passage. The uninvited visitor was a well-known—known, indeed, to his very finger-tips—but not an estimable character; at the same time he was prompt in resource and had the courage of his opinions. If he had the courage of all his convictions he must certainly have been a very daring person. He sprang instantly at the constable, and closed with him, jobbing at his face either with a chisel or with the chisel end of a jemmy. He then made off, but was immediately afterwards captured. The constable had but two or three clean cut wounds of the face, such as might have been caused by falling against broken glass; but he had been, and most unexpectedly, through a very trying experience. The wounds healed without any difficulty, and there was no other injury. After four months he returned to duty. Four years later he began to fail. Then mental symptoms supervened, and one day aphasia came on suddenly, while he was away in the country endeavouring to recruit his health. The cerebral symptoms grew worse, and he died some time after an operation for cerebral tumour. The possibility of a causal connection between the injury and the disease from which he died is rendered the more probable by the fact that I have met with at least two other cases which ran much the same course, and in which death was due to the same condition. Cases of the kind, indeed, are not infrequent. Such a train of symptoms it is fully recognised may follow after injuries in railway collisions, bad falls from horseback and the like, and will probably be frequently met with as the result of motor-car accidents. If the patients are wealthy enough to be able to purchase exemption from the strain and responsibilities of a working life,

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they may avoid progressive degeneration, though, to use the common expression, they "are never the same men again." To recapitulate ordinary cases of traumatic neurasthenia, now recognised as a very definite disorder, would be a profitless demand on your patience. But the essential feature of the type of cases I have in mind, and of which the examples mentioned are drawn from a fairly large group, is the comparative insignificance of the actual surgical injury. A good example was seen in a particularly intelligent, keen officer, who had risen rapidly from the ranks. In endeavouring to capture a man who had taken refuge in a room looking out into an area, the officer jumped up from the area and got his hands on to the window-sill, meaning to draw himself up. The man within struck at the officer's hands with a chopper and almost severed part of the little and ring fingers of one hand. Even then the officer made, very pluckily, further attempts to effect the arrest, for a genuine burglar is to a police officer what a "Royal" head is to the deer-stalker. Portions of the two fingers had to be amputated. There was no other injury of any kind. From the surgical point of view the case was trivial enough and the wounds healed readily. The hand was but little mutilated and was strong and useful enough for the work. In ten weeks the man returned to work apparently sound in mind and body, but within a few days the usual train of symptoms appeared, commencing as is almost invariably the case with insomnia broken by snatches of sleep, of a kind that Sir Arthur Helps would have designated by a dark shade of colour, and evil dreams. It seemed likely that complete breakdown would occur, and the officer was put on pension. Anxious to resume he returned to work nine months later and was sent to a healthy country district where the work was of an easy character. Some two months later, while walking along quietly with another man, he fell down insensible almost without warning and without apparent cause. A week or two later a similar attack occurred after a short railway journey. There were no symptoms of epilepsy on either occasion. Later on some optic neuritis developed, but passed off. Now two and a half years after the original injury he would be classed by some, but with rather a stretch of the significance of the term, as a "neurasthenic." Practically the wage-earning capacity is in such a case almost destroyed, and its recovery is very doubtful.

As an example of a type of case that is not at all uncommon in my experience in which the injury was still more trivial but the after-effects more grave, the following may be mentioned: A remarkably tall, powerful, stolid officer, of about thirty years of age, and nine years' experience in his duties, had occasion to arrest two men of the ordinary street loafer type, neither of them of any marked physique. The offenders were marched off one in each hand. After a while the men appeared suddenly to realise that their position was ignoble and that the odds were in their favour. By a rapid concerted action they made a bid for freedom, with the result that all three fell to the ground. In the scramble that ensued one of the men struck the constable a blow on the side of the neck with his fist, but this was practically the extent of the casualties. The officer held on and ultimately the group was re-formed and the procession continued as before. So trifling was the bruise that the officer, having disposed of his charges, went straight back to his duty. The period of "incubation" described by Charcot was well marked in this instance. For a few weeks all went well. Then, one day, the officer found that his head was drawn to one side, in a position of marked torticollis. It now transpired that mental symptoms had been insidiously coming on for some days previously. The officer's lot was cast in a rough neighbourhood, but he ran no more actual daily risk than he had been accustomed to for years. As usual, the symptoms took the form of delusions of persecution. First came ideas that he was being watched and pointed at. Then he imagined, especially when on night duty, that he was being followed. He fought for a while successfully against these delusions, and would have gone on struggling against an increasing mental degeneration had not the torticollis supervened. Two days after going on the sick list acute mania developed with strong suicidal tendencies. In two or three days he recovered from this attack and appeared absolutely well. The torticollis lasted only for a day or two, but a fortnight later he was in an asylum. After a stay of six weeks he recovered and has remained well since—three and a half years, though on the brink of further breakdown, and of course perma-

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nently unfitted for his former duties. If the injury to the neck had been received under more commonplace conditions, say in playing football, it is very difficult to believe that a similar train of symptoms would have developed. Matters may go even further. I can recall a case which happened within a few yards of where I was standing, full thirty years ago, and which first led me to realise the importance of the point as to causation that I am endeavouring to urge. The side of a large water tank on the roof of a hospital gave way. The initial weight of the mass of water and iron was computed subsequently at about five tons, but of course the water began to spread immediately. At any rate it burst through the roof into a ward immediately below on the second floor. It cut a clean square hole in the floor and carried away three beds with their occupants into the first floor ward; cut a similar hole through this floor and carried down three more beds on to the ground floor, where the six beds and the mass of rafters and *débris* finally came to rest. In a bed on the second floor was a woman who was convalescent from an operation for carcinoma of the breast, and who was about to leave the hospital. The sole visible injury that she sustained was a small scalp wound. She was seen within a few minutes of the accident, and there appeared to be no cerebral injury whatever. A day or two later she appeared perfectly well save that there was an almost unnatural calm of manner, such as is seen at times after severe burns and constitutes such an unfavourable feature. The late Mr. Cæsar Hawkins, whose ripe experience and thoughtful judgment constituted him one of the very foremost consultants of his day, was brought to see her. I can still recall his words and almost the measured emphatic inflection of his brief sentences: "The patient," he said, "has no doubt had a most marvellous escape. There seems no reason to suppose that she has sustained any injury beyond the scalp wound which we see. I think she will die. If you ask me for an explanation of this opinion I can hardly answer you. But my experience is that when persons receive an injury of this degree of gravity from a very great cause indeed they often die." The opinion was fully confirmed. In three weeks or so the patient faded out and died despite all that could be done for her. No obvious pathological explanation of the precise cause of death was forthcoming. Such an issue is no doubt an extreme one, but none the less significant as illustrating the factors that should always be taken into account.

In some of the cases cited the possibility of there being an element of malingering may occur to some, but none have been mentioned where even a suspicion of anything of the kind could not be wholly excluded. Exceptional opportunities are afforded in the class from whence my experience is drawn for ascertaining, so far as is humanly possible, the existence of any such factor. The men are commonly only anxious to resume their former occupation. They have everything to gain by doing so. They are debarred usually from resuming because they are public servants, and it is in the interests of the public that the work should be entrusted, so far as can be secured, only to those who are fully competent to discharge the duties. Other employment is hard, often impossible to get, for employers look askance at a servant who has been for any cause invalided out of any sort of occupation. Cases of the kind would be frequently enough met with in hospital practice were it possible to follow them up. If a full wage-earning capacity were taken as the test, the percentage of apparent complete recoveries would be much smaller than is imagined. It is difficult to say what becomes of these people. But few return to the hospitals, for they are not likely to be judged suitable cases for admission or even treatment. Probably most of them ultimately drift into those rich but wasted mines of clinical material, the infirmaries. There, to paraphrase Pope, unlamented they steal from the world and die, and no stone marks where they lie.

In most of the instances given the occupation entailed a considerable amount of responsibility which could in no degree be deputed to others, and demanded also the quality of initiative. The more purely mechanical the occupation the less will such after-effects, if they arise at all, be noticed. The ordinary manual labourer will, of course, be less prejudiced, and may be fully equal to such output of work as his trade union permits. The well-to-do person again can largely regulate his work and his responsibility according to the state of health. Recovery will go on at both extremes; but the man who has to struggle with increasing effort against a developing train of disabilities must

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sooner or later reach the breaking-point, and, too often, to anticipate is to destroy all chance of absolute recovery.

Allied to these is a class of cases unfortunately only too common, namely, injuries to the testicles. The hooligan has his own system of ju-jitsu, and a very favourite method of attack is kicking at his would-be captor's knee or testicles. That the latter form of injury is likely to give rise to serious mental effects is well known, and I have had only too frequent opportunities of watching the gravity and the permanence of such after-effects in cases in which all suspicion of malingering could be practically excluded. When a man following an occupation is anxious to return to it—when it is wholly to his own interest to resume that occupation—when he does so return to it, perhaps, on more than one occasion, and breaks down after a short time, and when a progressive mental degeneration steadily ensues, the question of malingering can be wholly discarded. The genuineness of symptoms such as intense neuralgic pains of the testicles, or even marked incontinence of urine, which I have known follow, may be questioned, but the psychical changes in men who can without difficulty be kept under close but unsuspected observation cannot be gainsaid. Here, again, the circumstances under which the original injury was sustained are of much moment, as also the degree of the responsibility attaching to the occupation. The after-effects cannot be estimated solely by the extent of the physical lesion. A man might meet with a similar injury in the hunting-field, and recover fully enough to resume his recreation with zest, as I have often known those who have met with grave accidents of all kinds in mountaineering pursue again their favourite pastime. But in such instances the element of responsibility is lacking. There are few more delicate tests of health than the weight of the responsibility a man can bear without appreciable effort or ill-effects to his physical or mental health.

Psychical after-effects are, of course, commonest after head injuries, but I pass them by in a few words. Such cases are more likely to be followed up, and, indeed, there is much more information to be obtained about these than about other apparently more trivial injuries. The factors to which allusion has already been made apply with increased force to this class of cases. The truth of time-honoured maxims, such as "No injury to the head is too trivial to be despised or too serious to be despaired of," is borne out by the experience of everyone, though with regard to apparently slight cases the lesson is still too often enforced by practical neglect of its wisdom. The value, indeed the essential necessity, of rest is recognised by all; it is agreed that the rest should at all times be protracted; it is less universally recognised that it must be prolonged in proportion to the degree of responsibility attaching to the patient's occupation and his normal mental capacity for bearing the same. These slight injuries—such as are constantly treated in the casualty or out-patient departments of hospitals for but a short period—I have exceptional opportunities of watching, from the first and over long periods of time. In the police force a wise provision enjoins that when any form of head injury has been sustained the man must be seen by his divisional surgeon. He is seen very shortly—often within a few minutes—after the injury. Minute reports are prepared and preserved. Long-continued rest, if necessary, can be secured. When the injured man resumes work he is, as at all times, under close supervision. Thus the persistence or the onset of even very slight mental changes will be noticed, for it does not need a medical man to recognise them. The facilities, therefore, for the close observation of the after-effects of slight head injuries are particularly favourable. In some excellent lectures delivered at the Royal College of Surgeons, Mr. Crisp English dealt with this subject as affecting hospital patients. Roughly, he found that in cases classed under cerebral concussion, contusion and laceration, without evident fracture, one half of the patients recovered and showed no after-effects. Ten per cent. showed mental impairment in a marked degree. My own experience is decidedly less favourable. Three factors have to be taken into account in explanation of this result. One is that the injury is usually sustained under trying and exciting conditions, intensified by the necessity for the exercise of strong control; secondly, the occupation is of a responsible nature; and lastly, it involves night work and solitary work. In the cases, too, that I have watched a large proportion have suffered from head injuries so apparently slight that, in hospital work, they might have escaped notice altogether. The sequence of

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symptoms is remarkably uniform. After a variable interval—determined more by the patient's temperament than the estimated degree of the surgical injury—the train of troubles begins with insomnia. Then follow lack of concentration, impairment of memory and most probably irritability. Such after-effects are in themselves gravely prejudicial in an occupation that at any moment may call for coolness and judgment in dealing with emergencies and in which accurate evidence may subsequently be required in Courts of Law or elsewhere. The public is highly critical in these matters and possibly a little selfish, but it rightly requires to be properly protected. So the individual has to be sacrificed to the general weal. As a distinguished French statesman expressed it, "L'État n'a pas d'entrailles." Of the long persistence of the mental impairment in injuries rather more severe in degree, a fair number come under observation or are to be found in the accurately prepared and carefully preserved records. A certain number of men who have had head injuries and apparently absolutely recovered have been allowed to return to the work after intervals of one, two, or rarely, more years. Often they desire only to put in short periods of duty with the view of completing service. An almost infinitesimal proportion succeed in achieving their object. Usually they break down within two or three months, sometimes in two or three days. The second convalescence, if it ensues at all, is likely to be more tedious and trying than the first. A man loses heart if he finds that his supposed recovery is but imaginary and that his powers only appear normal when he is inactive. This depression is serious, for, in most of these cases, mental after-effects are likely to pursue the direction of melancholia.

Of still graver injuries in which there is evidence of gross lesion of the brain and its membranes, there is little to be said. In the presence of progressive pathological degenerations the loss of mental powers, most probably tending in the direction of amentia, can be predicted and explained. The factors, of which the importance has been urged in the slighter injuries, are submerged in the grosser lesions. A single instance of the after-effects of a somewhat severe head injury may be quoted, partly on account of its almost dramatic interest and partly because the case illustrates most of the points to which attention has been drawn. The man was found lying in one of the parks in an unconscious condition. He was removed to hospital. The hospital record of the case is to the effect that the patient had concussion and that there was some cerebral irritation. He had two scalp wounds, one above, and the other just behind the right ear. In about twelve hours he recovered consciousness, though he was rather drowsy. He was unable to remember anything about the circumstances of the injury. A week later it was noted that he was rapidly recovering. There was some sub-conjunctival hæmorrhage which was not considered to be due to any fracture of the skull. The scalp wound had nearly healed. After a further fortnight he was regarded as convalescent and was allowed to sit up, and a week later—a month after admission—he was discharged. The nature of the wounds about the head from the first led to the conclusion that they had been caused by the buckle of a soldier's belt, and suspicion fell on men from a certain regiment. Shortly after the patient's admission, and when he was still dazed, though his consciousness was returning, he on one occasion made a spontaneous effort to recollect the circumstances of the assault. The only result of this effort was a murmured remark, made as it were to himself, to the effect, "I am trying to recollect" and then he named another regiment. The portals of memory, after thus standing ajar for a moment, closed again for good. The line, however, was followed up and the man was brought to trial and convicted. The assailant may be by now at liberty again, possibly somewhat chastened by his experience. It may be that laudable efforts are being made to reform him and to set him in the way of earning an honest living. And the victim? If only for the medical interest the after-history is worth recording. The victim of the assault—seven years after the injury—though physically in moderate health, is, mentally, a wreck, with constant headache, loss of memory, failing vision, and all the usual train of symptoms. Of the completeness of the breakdown and as evidence that the symptoms are genuine I may mention the fact that the man, being unable to look after himself, has to provide himself out of his own very limited means with constant attendance. His life is wrecked and will remain so. This is but an ordinary type of case in a man

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occupying a humble position in life, and is but one of many similar instances of what from the hospital treatment point of view might be tabulated as a recovery, but in which really the after-effects constitute the whole gravity and importance of the case.

This leads to a subject to which only the briefest allusion can here be made. Traumatic insanity, originating from head injuries, is held by some to be only an occasional sequel. It is true that there is much divergence of opinion on the question. One feature may be noted, namely, that, as in the small but still well-defined group of cases in which insanity follows surgical operations, so after head injuries the factor of heredity counts for little. Determination of the relative frequency of this grave after-result depends on the opportunity afforded for following up slight cases for a long period of time. In the class of men whom I have had mainly under consideration this is possible. Experience shows that after slight head injuries the actual occurrence of traumatic insanity is actually rare, but the liability to it is considerable. The factor of responsibility is the main cause. Not only is the man affected adversely by the consciousness that he has a responsible duty to discharge, but he feels also that failure to discharge it might by the committal of a single blunder injuriously affect a career and reputation laboriously built up, and which, though humble, are of vital importance to the individual. The double strain is too much for the weakened powers of judgment, volition, and control. So these men walk on the edge of a cliff, ever looking down and realising that the effects of a slip will be disastrous.

An eminent statesman has, in an address, dwelt on the charm of desultory reading. Few who have had the opportunity in a well-chosen library will gainsay its fascination even if they question the profit. But the desultory discussion of a comprehensive subject conventionally dealt with by elaborate analysis, statistical tables, and percentages may be but ill-suited to this occasion—one that, while it has recurred annually for much more than a century, has perhaps this year a special significance. For recently it appeared to be within the bounds of possibility that this annual custom might have come to an end had the fusion of kindred bodies—in another place—led to the partial submergence of this Society. However, it is past history that the Fellows decided to maintain intact its separate and individual existence. It is well that this should be so. When we glance back at the birth of an Association such as this we are apt to ascribe the origin too exclusively to those whom we honour as Founders. But while remembering the men let us not forget the circumstances that called them forth. This Society was a direct result, a crystallisation as it were, of the marked advance that took place in the development of scientific medicine and surgery towards the close of the eighteenth century. For those were the days of William and John Hunter, to mention but two conspicuous names amongst many who were prominent contributors to this phase of intellectual activity. The inception of the Medical Society may be regarded as the almost inevitable outcome of a definite advance along the whole line in scientific progress. As such it becomes a monument recording a great episode in the history of medicine, and a landmark to be by all means preserved. Still the Society could never have reached its present venerable age, while retaining all the characteristics of vigorous growth, had it not ever kept abreast with the times. Well has this essential condition always been fulfilled, for the records show that in all subsequent progress the Society has ever taken an active and honourable share. May it continue to follow the same lines—looking back with reverence to its past and with confidence to its future.

*October 5th*, 1908.

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**Epilepsy.**—Polk states that he cannot find a single course of treatment recommended by high authorities that has not been set aside as valueless by other authorities entitled to equal respect and credence. He insists that reflex epilepsy does exist, and holds that the denial of that fact by some causes many cases of reflex epilepsy to become permanent, when in earlier stages it would have yielded to correction of defects. The general treatment he does not discuss, as it is so various and disputed, and each practitioner will select for himself and be guided by his own results. The hygienic treatment he holds of equal importance with the medicinal, and he says that a course of treatment along comprehensive lines should be mapped out and the patient be required to give reports as to his adherence to it.—*Journ. A. M. A.*, vol. li, No. 7.

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# A CLINICAL LECTURE
ON
# A CASE OF BRONCHIECTASIS.

Delivered at St. Bartholomew's Hospital.

By J. A. ORMEROD, M.D., F.R.C.P.,
Physician to the Hospital, and to the National Hospital for the Paralysed and Epileptic, Queen Square.

GENTLEMEN, You know that one of the difficulties in hospital practice, with regard to chronic cases at any rate, is that you cannot always watch them through to their termination, because patients go first to one hospital, then to another, and in that way are often lost sight of. But there are occasional cases in which the course of the illness can be followed, and that is partly the reason for my choice of this case for to-day. The boy in question, who died when he was twelve years of age, must have spent a large part of his life in this hospital. He suffered from bronchiectasis. This term, used in a general way to signify dilatation of the bronchi, embraces a large variety of cases. At one end of the scale there is acute dilatation, which is multiple and affects both lungs, and generally occurs in children. At the other end there are the cases in which there is a single large cavity due to the dilatation of one bronchus. Between those two kinds there are various gradations. I do not intend to deal with all these, but shall content myself with saying that this particular case is one of a common type, in which one lung only is affected, and though in that lung many bronchi are dilated, yet there is one cavity sufficiently large to give rise to local physical signs. In this case, too, as is common, the dilatation of bronchi is associated with very great fibrosis of the affected lung. It will be worth while to sketch the usual symptoms of such a case, so as to avoid repetition later on. Generally there is a history of chronic lung disease, which may have had a definite starting-point in a pneumonia or pleurisy, or in an acute specific fever such as whooping-cough, measles, etc. The signs are largely those of unilateral consolidation of the lung, that is to say, dulness and bronchial breathing. But there may be also signs of excavation, pectoriloquy, and amphoric or cavernous breathing. Such signs, of course, are also common in pulmonary phthisis, but there they are usually found first at the apex of the lung; in bronchiectasis they are commoner in the lower lobe. Further, in cases of any standing there are almost always signs of shrinking of the affected lung, such as exposure of the heart, or displacement of that organ towards the affected side, an upward increase in the area of stomach resonance, and a shrinking of the whole of the affected side. In addition you may find such symptoms as clubbing of the fingers and of the toes. Just as in a case of phthisis, the patient is very liable to attacks of hectic fever. The character of the sputum is important. It is commonly muco-purulent, and often very foul smelling. It is usually copious in quantity, and it is loaded with micro-organisms, generally those of putrefaction, though not including tubercle bacilli. Sometimes it is mixed with blood, and there may be in some cases copious hæmoptysis. A very characteristic feature about the sputum of bronchiectasis is that it comes up in a paroxysmal way: the patient may have hours without any cough, but at the end of that time he will cough violently and bring up great quantities of this foul sputum, and then again have peace for the rest of the day. Thus the paroxysmal expectoration of copious foul sputum is very characteristic of bronchiectasis. I do not say that it is met with in no other disease, but certainly it is a common symptom of bronchiectasis.

The course of such cases is usually chronic, and the termination may come in various ways. I need not specify all these, but I may say in advance that this case terminated in abscess of the brain, an ending which is not, I think, very rare.

I will now ask you to follow the course of the case as I tell it to you. The boy first appeared at the hospital when he was five years of age. He was then admitted into Rahere Ward on March 11th, 1898, and went out on June 31st. His symptoms were particularly interesting. The history says he was always subject to bronchitis, being usually ill in the winter, and that he caught cold on the preceding March 5th and had cough and short breath, for which he was brought to the hospital. The striking point after his admission was his temperature, which oscillated widely: it was normal in the morning, and rose to 103° F. or 104° F. regularly in the evening. The apex beat was two inches outside the nipple line. There was a loud systolic murmur heard at both apex and base of the heart. He had loud rhonchi all

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over his lungs and impaired resonance with bronchial breathing at the base of the left lung. There was expectoration of blood and pus. The peculiar temperature, coupled with the signs of valvular disease, raised the suspicion of infective endocarditis, and it was resolved to administer anti-streptococcic serum. The fever had lasted for ten days when on March 21st he had a dose of anti-streptococcic serum, and the temperature then sank rather rapidly to normal, and remained so until April 4th. It then rose again, though not so high as 104° F., and this second rise lasted up to April 14th. He then had two or three more doses of anti-streptococcic serum, and the temperature again fell and remained more or less normal till his discharge from the ward. It was, therefore, a natural conclusion that the anti-streptococcic serum had done a great deal of good. He went out with the report that he was much better, but still had signs at the base of the left lung. He went to Swanley Convalescent Home. He came back to Rahere Ward on July 4th of the same year, and remained in the ward until August 10th, with similar signs in his lungs, the same murmur as before, and with a hectic temperature which lasted a week. This time I do not think any anti-streptococcic serum was given. Nothing more is known about him until September 29th, 1900, when he came into Pitcairn Ward to have a hernia cured. He was operated upon there for hernia. On October 24th apparently the wound had done well, but his temperature rose again and reached 103·4° F., and it remained high with occasional falls up to November 2nd. Apparently no special treatment was given. The signs in the lungs on this occasion appear to have been much the same as before. These facts raise a doubt as to the original diagnosis of infective endocarditis, and suggest that the attacks of fever were really connected with the state of the lung. For, in the first place, it is very rare for infective endocarditis to get well; and in the second place, after the boy had apparently got well the fever twice recurred, and he twice again recovered; whereas the disease of the lung was permanent, and septic absorption from it was a constant possibility. After his discharge from Pitcairn, it seems that he was re-admitted to the hospital on several occasions, but I have been unable to find the notes of these dates. The next date at which I can take up his history is when he came, at the age of twelve, as an out-patient to Dr. Fletcher. This was in January, 1905, and he remained under Dr. Fletcher's care for over two years. Dr. Fletcher kindly allows me to refer to his notes of this period. The physical signs did not vary much; there was, from the first, evidence of contraction of the left lung, and displacement of the heart to the left; and over the left lung, in front, impairment of resonance and of vocal vibrations; behind, impaired resonance throughout, bronchial breathing and some crepitation near the angle of the scapula. This dulness at the left base appears to have increased; and cavernous breathing, with pectoriloquy, at the angle of the left scapula is noted in May, 1906. The sputum was said to be copious, often foul, and streaked with blood (November, 1905). There was hæmoptysis in March, 1906. In March, 1907, he had pains in the legs and swelling of the joints, particularly of the left knee, and he was admitted to Matthew Ward, where he remained till October, 1907. Doubtless many of you may remember him there. The signs in his lungs remained as before, namely, impairment to percussion with feeble breathing sounds over the left upper lobe; dulness at the left base with bronchial, sometimes amphoric breathing, and consonating râles; these latter, however, varied according as the cavity in the lung was full or empty. The cardiac murmurs were still audible. His temperature often rose, but rarely above 100° F., or at most 101° F. At a certain time in the day he used to cough up a large quantity of greenish-yellow evil-smelling sputum. It contained no tubercle bacilli. The expectoration of this stuff was much facilitated by placing him with his head hanging down over the side of the bed, so that he was almost upside down. He was given creasote as a mixture with cod-liver oil; and as an inhalation, without much benefit. Finally we tried the plan which was recommended originally by Dr. Arnold Chapman, and which consists in the inhalation of creasote fumes in a closed chamber. He says in the 'Practitioner' of December, 1906, that a small room should be provided seven or eight feet square, sufficiently closed to prevent undue escape of vapour, but nevertheless allowing a certain access of fresh air. In the centre, on a raised stand, is placed an enamel dish, with a spirit lamp underneath it. Five or six ounces of commercial coal-tar creasote

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is poured into the dish, the lamp is lighted, and the creasote driven off in vapour. The patient, whose eyes should be protected with watch-glasses and nostrils with cotton-wool, is shut into the room and breathes the vapour. The sittings should be for twenty minutes a day, gradually increased until one hour is taken at a time. The room is made so dense with fumes that it is difficult to distinguish objects, but for the first few sittings the fumes are made less dense. The attendant should watch the proceedings through a piece of glass let into the door. The daily sittings should be continued from six weeks to three months. After that time they may be taken bi-weekly. The result is said to be the relief of the fœtor and the symptoms connected with it. In our new out-patient block, in the basement, baths have been constructed, and here there is a special chamber which can be used for this purpose. It has a window and a glass door, and a ventilating shaft with a valve worked from the outside, so that the fumes can be let out before the chamber door is opened. But for this boy a temporary chamber of the kind was made. The first effect of the inhalation on the patient is to produce a violent fit of coughing. That is good for him, because it helps to clear the sputum from his lungs. It is also thought that the creasote gets at the cavities themselves and brings about disinfection of them. This boy was treated in this way for three or four months. Though not cured, he was certainly benefited. He gained a good deal of flesh and strength. He did not lose his expectoration, but the fœtor of it disappeared. He did not altogether recover from his hectic fever, but he had long periods during which the temperature remained normal. Unfortunately, further developments were to come. He left the hospital in October, 1907, but subsequently he again came under Dr. Fletcher's care in the Out-patient Department on November 27th, 1907. On December 18th there was this note: that he had had headache, vomiting, and weakness of the right arm and leg, and now had on the right side paresis of the arm and leg, increased knee-jerk and ankle clonus. In consequence of these grave symptoms he was readmitted to Matthew Ward on December 28th, 1907. The mother gave this additional history: that a month before he had had considerable hæmoptysis, coughing up "a pint" of blood and phlegm, and from that time he commenced to have headaches. He had had another but slighter hæmoptysis fourteen days before admission. Further, that on December 15th, after an attack of coughing, he sat down, feeling faint and giddy, and his right hand dropped, and he complained of "pins and needles" in his right arm and leg. Since then he had had several similar attacks. On December 18th his mother noticed that, during one of these attacks, his right arm and leg muscles were twitching. The fingers did not move, and there was no movement of arm or leg as a whole, nor of the face. He never lost consciousness in these attacks. Now these symptoms—unilateral convulsions and unilateral paralysis—pointed to disease of the opposite cerebral hemisphere. And seeing that he had old bronchiectasis, the chances were that the nature of this disease was abscess; for abscess of the brain is a recognised result of bronchiectasis. I asked Mr. Bowlby to see him, and he did so on January 1st. At that time the patient seemed comfortable and intelligent, and there did not appear to be much wrong with him beyond the slight paralysis of the right side. But I saw him half an hour afterwards and he had become almost comatose. It was obvious that if anything was to be done it should be done at once. Mr. Bowlby not being then available, Mr. Bailey took the case in hand, and it was resolved to trephine him. The symptoms pointed to disease in the left motor area, and so Mr. Bailey trephined the left parietal bone over the arm centre. The dura mater was found to be very dense but pulsating. A director was passed into the brain substance in various directions in the hope of striking an abscess, but no pus was found. Three days later, January 4th, he had a fit, in which the left face twitched, followed by two others in which the left limbs also twitched. Next day (January 5th) the boy died, and on post-mortem examination it was found that the left lung showed great fibrosis and many dilatations of the bronchi, there being a large one in the position over which bronchial breathing and pectoriloquy had been heard during life. The other lung was normal. In the brain an abscess was found behind the fissure of Rolando, and not involving (or only slightly involving) the ascending parietal convolution. It lay in the upper half of the right hemisphere and came tolerably close to the surface both on the external and mesial aspects. Down-

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wards it extended to the corpus callosum. It measured, roughly speaking, one and a half inches in diameter, and contained thick greenish pus; its walls were fairly thick. The convolutions over it were stretched and the fissures were obliterated. The veins were engorged. So the abscess lay just behind the motor area, and behind the trephine hole, and unfortunately the director had missed it. It only remains to show you the specimen of the brain and of the lung, which I now ask you to examine.

*October 5th*, 1908.

**Scarlet Fever.**—Williams says that we do not know the chief site of the virus, and hence the chief source of infection; or how long the virus may remain infective after separation from the patient; or the exact period of incubation; or any real pathognomonic symptoms, for the rash is seen in other cases—*e.g.* after serum inoculation; desquamation may occur after any erythema, and the enlargement of papillæ on the tongue, which is regarded as pathognomonic, is often so slight as to be difficult to make out, and, moreover, it has been reported as occurring in other diseases. The discussion over Duke's "fourth disease" demonstrates the uncertainty. Little work has been done on the minute pathology. The only constant change is a hyperplasia of lymphatic tissue over the whole body. The only characteristic change is the bodies found by Mallory in 1903, and these have so far received but little corroboration. Field has examined by an improved technique skin from 20 living scarlet fever patients and from 10 autopsies, with a number of controls, and has come to the conclusion that most, if not all, the bodies are degenerations of the cytoplasm of the host cells. None were found in living skin. Williams has completed an examination of skins from 17 living patients and 33 autopsies, with 9 controls. She reports the findings and states that so far there is no evidence that they are living organisms; neither has she been able to demonstrate anything like a tiny organism within the larger bodies. She cannot, therefore, agree with Prowazek in including scarlet fever in the group of diseases produced by what he calls "chlamydozoa."—*Journ. A. M. A.*, vol. li, No. 7.

# THE PERSONAL FACTOR IN ANÆSTHETICS.

By A. de PRENDERVILLE, M.R.C.S., L.R.C.P.,
Senior Anæsthetist to the London Throat Hospital, W.; Lecturer and Teacher of Anæsthetics to the North-East London Post-graduate College.

In view of the fact that physiologists have entered a very strong plea for the general adoption of anæsthetic machines, whereby dosage may be regulated and controlled, it may not be inopportune to insist *per contra* on the claim of the individual, *i. e.* the personal administrator, to that consideration which in all cases, and at all times, is unquestionably his due. Methods that are clearly based on scientific data must receive due and adequate attention, and though much that has been advanced has not gained universal approval, we cannot resist the obvious conclusion that automatic mechanism, in so far as the exhibition of chloroform is concerned, is not to be discouraged. How far the apparatus at present in vogue—and I refer to many modifications of an original theory—fulfil their avowed purpose is open to question. Under any circumstances it must be admitted that finality has not been reached, and anæsthetists as a body are satisfied that the term of their personal dominion is not yet at hand.

Surgical energy has been so marked in recent years that anæsthesia, once largely empirical, is now fixed on such a basis that we can with confidence lay down distinct and positive axioms. Practice, after all, teaches the real thing, and no amount of theoretic deduction in the laboratory can gainsay the truths evolved on the table and at the bedside. If one thing more than another has been established during the experience of the last fifty years it is this—that the personal equation counts for very much in the conduct of an anæsthetic sequence. For what, after all, is any craftsman at the zenith of his power? He is the trained man, built up step by step, tried in the crises of sudden alarms, not once alone but oft repeated, quick to observe each altering sign, prompt to act when action means everything; he is a complex product, into whose being many diverse factors have become welded to make a composite whole. But he is more than this. He is the skilled artist who does things well reflexly,

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without strain, with certain aim, always with anxious thought, unobtrusive in observation, calm, yet prompt in deed, admirable as helper in time of stress. He it is who has the *mens æqua in arduis*, that happy blend of grit and knowledge which just keeps the mental ballast poised midway.

To talk of reaction after labour for such an one is but to state a truism. See him in action, and you will then appreciate how finely tuned he is. Not even the surgeon, merged in the depths of adherent bowel coils or grappling with a brittle artery, has greater work to do; for he who holds the helm must respond with will and power to the demands made upon him by the changing needs of the operation, and oftentimes the urgent claims of the patient. He is in very fact as much a part of any surgical procedure as the surgical procedure itself. And if this be a bold thing to state, let me add that, in my view, the time is ripe for the forceful reiteration of facts, now and then forgotten in the domain of general surgery.

Years ago, when strong men took up the dangerous task of pioneering a safe way for anæsthesia out of the chaotic disorder that then surrounded the whole question, surgery itself had but a limited range of action. Specialism had not arrived, and Lister, whose advent changed the whole outlook, was still busy working out his epoch-making discoveries. With Antisepsis began the momentous change that brought, not only a complete revolution of methods and technique, but created new spheres of action hitherto undreamt of in the art. On all sides men availed themselves of the new dispensation, and with a bound an era of surgical activity destined to know no check was launched upon the civilised world. Simpson and those who immediately followed him, were content to leave Chloroform almost as they found it—highly useful as a narcotic agent, often dangerous, and physiologically a very dark horse indeed. No one seemed to know its intimate action on the heart or respiratory centres, yet all were agreed that it was an inestimable boon, and must remain. Now, curiously enough, while surgery was, so to speak, rising on "stepping stones of its dead self to higher things," anæsthesia made no move. For years crude efforts to keep pace with increasing demands were made, but they were merely spasmodic, and led to nothing. Anyone could hold a mask and pour on the drug—the larger the dose the better! Students and nurses, and in country places gardeners and even grooms, were often pressed into service. So things went on, haphazard truly and unscientific, but surely forging ahead.

The mere increase of work alone gave scope to investigators to pursue their studies with ease and accuracy; the material was abundant, the opportunities many. Yet with it all but slow progress followed. Until quite recent years, so recent indeed that one might speak of it as in our own time, anæsthesia *per se*, or, shall I rather say, the *rôle* of the administrator, had so little weight attached to it that we look in vain for records of a single special official in this branch, where now perhaps even four or five experts barely suffice to carry through the routine service of a hospital. Doubtless what impelled attention was, first, the wholly inadequate work of men picked at random and always fresh to the task, and secondly, the establishment of specialism in surgery.

The pioneers banded themselves together, working with one object and to one end, namely, the founding of a corps, carefully and accurately trained on special lines and for special service. Then, slowly but surely, the feeling gained ground, among operators chiefly, that pure anæsthetists were at hand; hospital appointments speedily followed, and so great became the demand that the supply was insufficient to fill posts newly created in every public institution throughout the country.

The anæsthetist, as a distinct and separate type of the profession, came upon the scene when specialism was just born. He was created by the exigencies of modern surgical life. Before his time general practitioners and students, qualified or unqualified, did all that was asked of them, but as time grew the labours imposed upon administrators became more arduous and exacting. Men were needed to devote their time and attention solely in one direction. They were called with no uncertain voice, and at once proceeded to justify their position, and to advance by very brilliant efforts the cause of scientific narcosis. I doubt much if any art destined for the relief of human pain has a worthier record than ours.

Looking back on the last twenty years we can see two things, therefore, prominent before us—the growth of specialism and of anæsthesia. And it is in this connection that I would make a strong

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claim for the clearer recognition of the personal factor in special operative work. Summoned by an imperative demand the anæsthetist expects, and should receive, a full measure of support. He was, until very recent years, the indispensable right-hand man of the surgeon, without whom, indeed, difficult and often dangerous work could not be undertaken. Special courses in hospital training are now given to students with the hope and expectation that they will be of service in practice. They give but little help in reality, and certainly are no guarantee of proficiency for the thousand and one details needed in special anæsthetic manœuvres. Nowadays, however, it is no uncommon thing to find work of a really difficult character entrusted to administrators who are not specialists, and who cannot therefore be expected to do full justice either to themselves or to the patient.

This question is likely to receive much closer attention in the near future, both at the hands of the profession and of the general public. Fatalities have unfortunately been more numerous of late in well-ordered places than the immunity hitherto enjoyed had led us to expect. Moreover, if coroners have power to hold inquests on all deaths following regular operations, we shall at once be face to face with another and a very grave factor in this connection, and that is, the vexed and difficult question of responsibility.

Here, obviously, the personal equation is clearly in view and cannot be put aside. Judicial inquiry probes deep, and the Press is not always as well in hand as it ought to be when topics of this character come up for criticism. One result will certainly ensue in the event of post-mortem publicity—if people submit to operation at all, they will exact guarantees of extreme efficiency from all concerned beforehand, and so place themselves, as far as possible, in a safe position. But apart altogether from the fears of disaster and the after consequences to the practitioner, is it not reasonable to demand that anæsthetic work should be distributed in anæsthetic channels? In this way alone can confidence be restored and the *status quo ante* re-established. Quite recently, as we all know, the columns of a leading daily paper were given over to the discussion of certain aspects of this and cognate questions, which can only help to foster still more the spirit of unrest Not only is the surgeon threatened by this new move—and, be it said, most unjustly threatened—but all along the line the profession will be made to feel the lash. There never was a time when greater individual efforts were called for and the services of the expert more urgently needed.

If I speak as a special pleader for anæsthesia, it is because I have some knowledge of its wants and claims; because I know that it has a mission in which the personal factor, despite anything that may be said to the contrary, must inevitably prevail.

*October 5th*, 1908.

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**Phlebitis Following Unoperated Appendicitis.**—Buhlig reports a case of so-called phlebitis of the left leg in a patient with appendicitis not operated on, beginning about the tenth day of the illness. The onset of the complication and its clinical features tally so exactly with those of many of the post-operative cases that, in Buhlig's opinion, they are of like nature. He then compares the possible factors present in this case with those suggested in the numerous theories that have been advanced in explanation of post-operative phlebitis. All relating to the actual operation and preliminaries can, of course, be discarded; also numerous factors —heavy bandages, sigmoid pressure, tumour, etc.— producing local or general abdominal pressure. Even the hot water bag was eschewed for fear of disturbing limiting adhesions. There had been no previous exhausting disease, no demonstrable feeble circulation and no anæmia. Buhlig further cites Welch's observation, in discussing venous thrombosis of the heart, that the small liability to thrombosis under the conditions of heart disease (in which slow, feeble and irregular venous current would be the paramount factor) is one of many evidences that mere slowing of the blood current is not an efficient cause. Ruling out any undetermined changes in the blood state with a subject still in a problematic stage, the chief factor remaining is any infection which might produce changes in the vessel. He considers infection the cause, and cannot see how appendicectomies and hysterectomies can possibly be made strictly aseptic. Of the infectious nature of true phlegmasia alba dolens, Buhlig says, there can certainly be no question.—*Journ. A. M. A.*, vol. li, No. 8.

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